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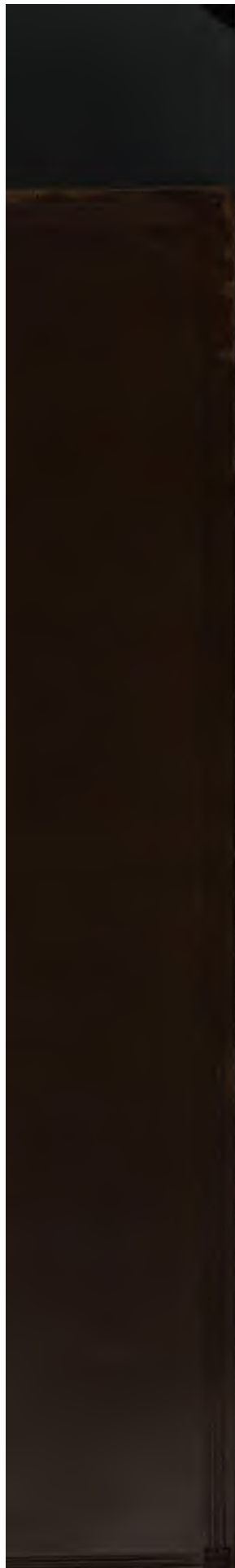
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MEDICO-CHIRURGICAL
TRANSACTIONS.

PUBLISHED BY

THE ROYAL
MEDICAL AND CHIRURGICAL SOCIETY

OF

LONDON.

VOLUME THE THIRTY-NINTH.

LONDON :
LONGMAN, BROWN, GREEN, AND LONGMANS,
PATERNOSTER-ROW.

1856.

MEDICO-CHIRURGICAL TRANSACTIONS.

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PUBLISHED BY

THE ROYAL
MEDICAL AND CHIRURGICAL SOCIETY
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LONDON.

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VOLUME THE TWENTY-FIRST.



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- 1851. JOSEPH HODGSON.
- 1853. JAMES COPLAND, M.D.
- 1855. CÆSAR HENRY HAWKINS.

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T.—Treasurer.	S.—Secretary.
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OCTOBER 1856.

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Elected

- 1841 *JAMES ABERCROMBIE, M.D., Cape of Good Hope.
- 1846 *JOHN ABERCROMBIE, M.D., Physician to the General Dispensary, Cheltenham; Cheltenham.
- 1851 *HENRY WENTWORTH ACLAND, M.D. F.R.S., Physician to the Radcliffe Infirmary, Oxford.
- 1842 WILLIAM ACTON, 46, Queen Anne-street, Cavendish-square.
- 1818 WALTER ADAM, M.D., Physician to the Royal Public Dispensary, Edinburgh.
- 1851 JOHN ADAMS, Surgeon to the London Hospital; 4, St. Helen's-place, Bishopsgate-street.
- 1852 WILLIAM ADAMS, Assistant-Surgeon to the Royal Orthopædic Hospital; Lecturer on Surgery at the Grosvenor-Place School of Anatomy; 5, Henrietta-street, Cavendish-sq.
- 1818 THOMAS ADDISON, M.D., Physician to, and Lecturer on Medicine at, Guy's Hospital; 24, New-street, Spring-gardens. C. 1826. V.P. 1837. P. 1849-50. C. 1853.
- 1814 JOSEPH AGEH, M.D., 85, Great Portland-street, Portland-place. C. 1836.

Elected

- 1837 *RALPH FAWSETT AINSWORTH, M.D., Manchester.
- 1819 GEORGE FREDERICK ALBERT.
- 1839 RUTHERFORD ALCOCK, K.C.T. K.T.S., China.
- 1826 JAMES ALDERSON, M.D. F.R.S., Physician to, and Lecturer on Clinical Medicine at, St. Mary's Hospital; 17, Berkeley-square. S. 1829. C. 1848. T. 1849. V.P. 1852-3.
- 1843 CHARLES JAMES BERRIDGE ALDIS, M.D., Medical Officer of Health for the Parish of St. George, Hanover-square; Senior Physician to the Surrey Dispensary; and Physician to the St. Paul and St. Barnabas Dispensary; 1, Chester-terrace, Chester-square.
- 1850 CHARLES REVANS ALEXANDER, Assistant-Surgeon to the Royal Infirmary for Diseases of the Eye; 6, Cork-street, Bond-street.
- 1813 HENRY ALEXANDER, F.R.S., Surgeon-Oculist in Ordinary to the Queen, and Surgeon to the Royal Infirmary for Diseases of the Eye; 6, Cork-street, Bond-street. C. 1822, 1840. V.P. 1850.
- 1836 HENRY ANCELL, 3, Norfolk-crescent, Oxford-sq. C. 1847-8.
- 1817 ALEXANDER ANDERSON.
- 1820 THOMAS ANDREWS, M.D., Norfolk, Virginia.
- 1813 WILLIAM ANKERS, Knutsford.
- 1819 PROFESSOR ANTONMARCHI, Florence.
- 1819 JAMES MONCRIEFF ARNOTT, F.R.S., 2, New Burlington-street. L. 1826-8. V.P. 1832-3. T. 1835-40. C. 1846, 1855-6. P. 1847-8.
- 1817 JOHN ASHBURNER, M.D., F.L.S., 7, Hyde Park-place, Cumberland-gate. C. 1821, 1830-1.
- 1851 THOMAS JOHN ASHTON, Surgeon to the Blenheim Dispensary; 31, Cavendish-square.
- 1825 BENJAMIN GUY BABINGTON, M.D. F.R.S., Physician to the Deaf and Dumb Institution; 31, George-street, Hanover-square. C. 1829. V.P. 1845-6. T. 1848.
- 1846 CORNELIUS METCALFE STUART BABINGTON, L.R.C.P., Physician to Queen Charlotte's Lying-in Hospital, and Assistant-Physician to the Hospital for Sick Children; 29, Hertford-street, May-fair.
- 1820 *JOHN H. BADLEY, Dudley.

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- 1840 WILLIAM BAINBRIDGE, Kingston, Surrey.
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- 1851 *ALFRED BAKER, Surgeon to the General Hospital, Birmingham.
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- 1848 EDWARD BALLARD, M.D., Lecturer on the Practice of Medicine at the Grosvenor-Place School of Medicine, and Medical Officer of Health for Islington; 42, Myddleton-square.
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- 1847 ANDREW WHYTE BARCLAY, M.D., Physician to the Chelsea Dispensary; Medical Officer of Health for Chelsea; 23A, Bruton-street, Berkeley-square.
- 1848 EDGAR BARKER, 9, Oxford-square, Hyde-park.
- 1833 THOMAS ALFRED BARKER, M.D., Physician to, and Lecturer on Medicine at, St. Thomas's Hospital; 71, Grosvenor-street. C. 1844-5. V.P. 1853-4.
- 1843 THOMAS HERBERT BARKER, M.D., Priory-terrace, Bedford.
- 1847 GEORGE HILARO BARLOW, M.D., Physician to Guy's Hospital; 5, Union-street, Southwark.
- 1840 BENJAMIN BARROW, Ryde, Isle of Wight.
- 1844 WILLIAM RICHARD BASHAM, M.D., Physician to, and Lecturer on Medicine at, the Westminster Hospital; 17, Chester-street, Grosvenor-place. S. 1852-4.
- 1841 GEORGE BEAMAN, M.D., 32, King-street, Covent-garden.
- 1856 AMOS BEARDSLEY, Ulverstone, Lancashire.
- 1836 WILLIAM BEAUMONT, Professor of Surgery in the University of King's College, Toronto, Upper Canada.
- 1840 CHARLES BEEVOR, 41, Upper Harley-street.
- 1819 THOMAS BELL, F.R.S. F.L.S., Professor of Zoology in King's College, London; Lecturer on Diseases of the Teeth at Guy's Hospital; and President of the Linnean Society; 17, New Broad-street, City. C. 1832-3. V.P. 1854.

Elected

- 1847 JAMES HENRY BENNET, M.D., Physician-Accoucheur to the Royal Free Hospital; 60, Grosvenor-street.
- 1845 EDWIN UNWIN BERRY, 7, James-street, Covent-garden.
- 1820 STEPHEN BERTIN, Paris.
- 1815 ARCHIBALD BILLING, M.D., late Senior Physician to the London Hospital; Examiner in Medicine at the University of London; 6, Grosvenor-gate. C. 1825. V.P. 1828-9.
- 1827 WILLIAM BIRCH, Barton, Lichfield.
- 1850 JAMES BIRD, M.D., Lecturer on Military Surgery at St. Mary's Hospital Medical School; 27, Hyde Park-square.
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- 1851 GEORGE BIRKETT, M.D., 1, Gloucester-villas, Highbury New Park.
- 1851 JOHN BIRKETT, F.L.S., *Librarian*, Surgeon to, and Lecturer on Surgery at, Guy's Hospital; 59, Green-street, Grosvenor-square.
- 1846 HUGH BIRT, Surgeon to the Morro Velhio Hospital.
- 1843 PATRICK BLACK, M.D., Assistant-Physician to, and Lecturer on Forensic Medicine at, St. Bartholomew's Hospital; Queen Anne-street. C. 1856.
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- 1847 GEORGE C. BLACKMAN, M.D., New York, U.S.
- 1839 RICHARD BLAGDEN, Surgeon-Accoucheur, and Surgeon Extraordinary to the Queen, Surgeon in Ordinary to H.R.H. the Duchess of Kent; 7, Percy-place, Walcot, Bath. C. 1847-8.
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- 1845 HENRY BLENKINSOP, Warwick.
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- 1816 HUGH BONE, M.D., Inspector-Gen. of Hospitals; Edinburgh.
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- 1846 PETER BOSSEY, Woolwich.
- 1846 JOHN ASHTON BOSTOCK, Battalion Surgeon, Scots Fusilier Guards; 34, Clarges-street, Piccadilly.

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- 1849 EDWARD BARONS BOWMAN, M.D., 4, Oxford-terrace, Middleton-road, Dalston; and Archerfield House, Highbury New Park.
- 1841 WILLIAM BOWMAN, F.R.S., Surgeon to King's College Hospital, and to the Royal Ophthalmic Hospital, Moorfields; 5, Clifford-street, Bond-street. C. 1852-3.
- 1814 RICHARD BRIGHT, M.D. F.R.S. D.C.L., Physician Extraordinary to the Queen, and Consulting Physician to Guy's Hospital; 11, Savile-row, Burlington-gardens. C. 1821-2, 1830, 1833, 1839-40. V.P. 1827-8, 1831-2. P. 1837-8.
- 1851 BERNARD EDWARD BRODHURST, Assistant-Surgeon to the Royal Orthopædic Hospital; 14, Brook-street, Grosvenor-square.
- 1813 SIR BENJAMIN COLLINS BRODIE, Bart., D.C.L. F.R.S., Serjeant-Surgeon to the Queen, Surgeon in Ordinary to H.R.H. Prince Albert, Foreign Correspondent of the Institute of France, and Foreign Associate of the Royal Academy of Medicine of Paris; 14, Savile-row, Burlington-gardens. C. 1814-5, 1818-9, 1821-2, 1835-6, 1841-2, 1849. V.P. 1816-7. P. 1839-40.
- 1844 CHARLES BROOKE, M.A. (Cantab.) F.R.S., Surgeon to, and Lecturer on Surgery at, the Westminster Hospital; 29, Keppel-street, Russell-square. C. 1855.
- 1848 WILLIAM PHILPOT BROOKES, M.D., Surgeon to the Cheltenham General Hospital and Dispensary, and Visiting Medical Officer to the Cheltenham District of Lunatic Asylums; Albion House, Cheltenham.
- 1842 CHARLES BLAKELY BROWN, M.B., Physician to Queen Charlotte's Lying-in Hospital, and to the St. George's and St. James's Dispensary; 38, Hill-street, Berkeley-square.
- 1847 GEORGE BROWN, Surgeon-Major, Grenadier Guards; the Hospital, Rochester-row, Westminster.
- 1854 *HENRY BROWN, Surgeon to the Queen, H.R.H. Prince Albert, and to the Royal Household; Windsor.
- 1847 *ROBERT BROWN, Winckley-square, Preston, Lancashire.
- 1851 ALEXANDER BROWNE, M.D., Army and Navy Club, St. James's-square; and Twynholm, Kircudbright.
- 1827 M. PIERRE BRULATOUR, Surgeon to the Hospital; Bordeaux.

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- 1855 WALTER JOHN BRYANT, 7, Bathurst-street, Hyde-park-gardens.
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- 1843 JOHN CHARLES BUCKNILL, M.B., Axminster, Devonshire.
- 1839 GEORGE BUDD, M.D. F.R.S., Professor of Medicine in King's College, London; Physician to King's College Hospital; Consulting Physician to the Blenheim Free Dispensary; 20, Dover-street, Piccadilly. C. 1846-7.
- 1839 THOMAS HENRY BURGESS, M.D., Military Hospital, Portsmouth.
- 1853 PATRICK BURKE, 13, Upper Montagu-street, Montagu-square.
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- 1833 GEORGE BURROWS, M.D. F.R.S., Physician to, and Lecturer on Medicine at, St. Bartholomew's Hospital; 18, Cavendish-square. C. 1839-40. T. 1845-7. V.P. 1849-50.
- 1837 GEORGE BUSK, F.R.S. F.L.S., Professor of Comparative Anatomy at the Royal College of Surgeons; Surgeon to the Seamen's Hospital Ship 'Dreadnought;' 15, Harley-street, Cavendish-square. C. 1847-8. V.P. 1855.
- 1818 JOHN BUTTER, M.D. F.R.S. F.L.S., Consulting Physician to the Plymouth Eye Infirmary; Plymouth.
- 1851 *WILLIAM CADGE, Assistant-Surgeon to the Norfolk and Norwich Hospital; All Saints, Norwich.
- 1851 THOMAS CALLAWAY, Assistant-Surgeon to Guy's Hospital; 7, Wellington-street, Southwark.
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- 1845 SAMUEL CARTWRIGHT, Jun., Surgeon-Dentist to King's College Hospital, 32, Old Burlington-street.

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 1844 THOMAS KING CHAMBERS, M.D., Physician to, and Lecturer on Medicine at, St. Mary's Hospital; 1, Hill-street, Berkeley-square.
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 1837 HENRY THOMAS CHAPMAN, 16, Lower Seymour-street, Portman-square.
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 1839 FREDERICK LE GROS CLARK, *Vice-President*; Surgeon to, and Lecturer on Surgical Anatomy at, St. Thomas's Hospital; Consulting Surgeon to the Western General Dispensary, and to the London Female Penitentiary, Pentonville; 24, Spring-gardens. S. 1847-9. V.P. 1855.
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 1845 JOHN CLARK, M.D., Staff Surgeon, 1st class.
 1848 JOHN CLARKE, L.R.C.P., Physician to the British Lying-in Hospital; 42, Hertford-street, May-fair.
 1850 JOSIAH CLARKSON, Birmingham.
 1842 OSCAR MOORE PASSEY CLAYTON, 87, Harley-street.
 1853 JOSEPH T. CLOVER, 44, Mortimer-street, Cavendish-square.

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- 1820 BENJAMIN COOPER, Stamford.
- 1819 GEORGE COOPER, Brentford, Middlesex.
- 1841 GEORGE LEWIS COOPER, Surgeon to the Bloomsbury Dispensary ; 7, Woburn-place, Russell-square.
- 1843 WILLIAM WHITE COOPER, Senior Surgeon to the North London Eye Infirmary, and to the Honorable Artillery Company, and Ophthalmic Surgeon to St. Mary's Hospital ; 19, Berkeley-square.
- 1854 CHARLES THOMAS COOTE, M.D., 1, Gloucester-pl., Hyde-park.
- 1841 HOLMES COOTE, Assistant-Surgeon to St. Bartholomew's Hospital ; S. 1853-4.
- 1835 GEORGE FORD COPELAND, Cheltenham.
- 1822 JAMES COPLAND, M.D. F.R.S., Consulting Physician to the Royal Infirmary for Children ; Hon. Fellow of the Royal Academy of Sciences of Sweden, &c. ; 5, Old Burlington-street. C. 1831. V.P. 1838-9. P. 1853-4.
- 1847 JOHN ROSE CORMACK, M.D.
- 1839 *CHARLES CÆSAR CORSELLIS, M.D., Resident Physician to the Lunatic Asylum, Wakefield, Yorkshire.
- 1853 WILLIAM GILLETT CORY, M.D., Sutton, Surrey.
- 1847 RICHARD PAYNE COTTON, M.D., Physician to the Hospital for Consumption and Diseases of the Chest ; 46, Clarges-street, Piccadilly.
- 1828 WILLIAM COULSON, Consulting Surgeon to the City Lying-in Hospital, and Senior Surgeon to, and Lecturer on Surgery at, St. Mary's Hospital ; 2, Frederick's-place, Old Jewry. C. 1831. L. 1832-7. V.P. 1851-2.
- 1817 *SIR PHILIP CRAMPTON, Bart., F.R.S., Surgeon-General to the Forces in Ireland ; Dublin.
- 1841 MERVYN ARCHDALL NOTT CRAWFORD, M.D., Wiesbaden ; C. 1853-4.

Elected

- 1847 GEORGE CRITCHETT, Surgeon to the London Hospital, and the Royal London Ophthalmic Hospital; 46, Finsbury-square.
- 1837 JOHN FARRAR CROOKES, Harewill, near Faversham, Kent.
- 1849 *WILLIAM EDWARD CROWFOOT, Beccles, Suffolk.
- 1818 WILLIAM CUMING, M.D., Professor of Botany at the Glasgow Institution, and Surgeon to the Royal Infirmary at Glasgow.
- 1851 JAMES CAMERON CUMMING, M.D., 1, Cadogan-place, Sloane-street.
- 1846 HENRY CURLING, Surgeon to the Royal Sea Bathing Infirmary; Ramsgate, Kent.
- 1837 THOMAS BLIZARD CURLING, F.R.S., *Treasurer*; Surgeon to, and Lecturer on Surgery at, the London Hospital; 39, Grosvenor-street. S. 1845-6. C. 1850. T. 1854-5.
- 1847 JOHN EDMUND CURREY, M.D., Lismore, Ireland.
- 1836 GEORGE CURSHAM, M.D., *Treasurer*; Physician to the Hospital for Consumption and Diseases of the Chest, and to the Female Orphan Asylum; 5, Savile-row, Burlington-gardens. S. 1842-7. C. 1850-1. V.P. 1855.
- 1822 CHRISTOPHER JOHN CUSACK, Chateau d'Eu, France.
- 1852 THOMAS CUTLER, M.D., Physician to the Spa General Dispensary; Spa, Belgium.
- 1828 ADOLPHE DALMAS, M.D., Paris.
- 1836 *JAMES STOCK DANIEL, Ramsgate.
- 1850 JOHN BAMPFYLDE DANIELL, M.D., Oriental Club.
- 1820 GEORGE DARLING, M.D., 6, Russell-square. C. 1841-2.
- 1818 *SIR FRANCIS SACHEVEREL DARWIN, Knt., M.D., Breadsall Priory, near Derby.
- 1848 HENRY DAUBENY, 40, York-place, Portman-square.
- 1846 FREDERICK DAVIES, 19, Upper Gower-street, Bedford-square.
- 1818 HENRY DAVIES, M.D., 6, Duchess-street, Portland-place. C. 1827-8. V.P. 1848-9.
- 1847 JOHN DAVIES, M.D., Physician to the Hertford Infirmary, and Visiting Physician to the County Gaol and Lunatic Asylum, Hertford.
1853. ROBERT COKE NASH DAVIES, Winchelsea, Sussex.

Elected

- 1852 WILLIAM DAVIES, M.D., Senior Physician to the United Hospital, Bath; Gay-street, Bath.
- 1852 JOHN HALL DAVIS, M.D., Physician to the Royal Maternity Charity; and Physician-Accoucheur to the St. George's and St. James's Dispensary; 11, Harley-street, Cavendish-square.
- 1820 THOMAS DAVIS, 28, Spring-gardens. C. 1837, 1843.
- 1818 JAMES DAWSON, Liverpool.
- 1847 GEORGE EDWARD DAY, M.D., F.R.S., Chandos Professor of Medicine, St. Andrew's.
- 1846 *SAMUEL BEST DENTON, Ivy Lodge, Hornsea, East Riding, Yorkshire.
- 1844 ROBERT DICKSON, M.D., Physician to the Scottish Hospital; 16, Hertford-street, May-fair.
- 1839 JAMES DIXON, Surgeon to the Royal London Ophthalmic Hospital; 45, Green-street, Park-lane. L. 1849-55.
- 1845 JOHN DODD.
- 1853 ROBERT DRUITT, L.R.C.P., Medical Officer of Health for the Parish of St. George, Hanover-square; 39A, Curzon-street, May-fair.
- 1846 JOHN DRUMMOND, Deputy-Inspector of Fleets and Hospitals; Royal Naval Hospital, Chatham.
- 1843 THOMAS JONES DRURY, M.D., Physician to the Salop Infirmary; Shrewsbury.
- 1845 GEORGE DUFF, M.D., Prospect Lodge, Elgin.
- 1845 EDWARD WILLSON DUFFIN, 14, Langham-place.
- 1833 ROBERT DUNN, 31, Norfolk-street, Strand. C. 1845-6.
- 1843 CHRISTOPHER MERCER DURRANT, M.D., Physician to the East Suffolk and Ipswich Hospital; Ipswich, Suffolk.
- 1839 HENRY SUMNER DYER, M.D., 37, Bryanstone-sq. C. 1854-5.
- 1836 JAMES WILLIAM EARLE, Norwich.
- 1854 BOOTH EDDISON, Surgeon to the General Hospital, near Nottingham.
- 1853 *GEORGE EDWARDES, Wolverhampton.
- 1824 GEORGE EDWARDS.
- 1823 CHARLES CHANDLER EGERTON, Kendall Lodge, Epping.
- 1848 GEORGE VINER ELLIS, Professor of Anatomy in University College, London.
- 1854 *JAMES ELLISON, M.D., Windsor.

Elected

- 1835 WILLIAM ENGLAND, M.D., Wisbeach, Cambridgeshire.
- 1842 JOHN ERICHSEN, Professor of Surgery in University College, London, and Surgeon to University College Hospital: 48, Welbeck-street, Cavendish-square. C. 1855-6.
- 1815 *GRIFFITH FRANCIS DORSETT EVANS, M.D., High-street, Bedford. C. 1838.
- 1836 GEORGE FABIAN EVANS, M.D., Physician to the General Hospital, Birmingham.
- 1845 WILLIAM JULIAN EVANS, M.D.
- 1844 ARTHUR FARRE, M.D. F.R.S., Professor of Midwifery in King's College, London, and Physician, for the Diseases of Women and Children, to King's College Hospital; 12, Hertford-street, May-fair.
- 1831 ROBERT FERGUSON, M.D., Physician-Accoucheur to the Queen, Physician to the Westminster Lying-in Hospital; 125, Park-street, Grosvenor-square. C. 1839. V.P. 1847.
- 1841 WILLIAM FERGUSON, F.R.S., Surgeon Extraordinary to the Queen; Surgeon to H.R.H. Prince Albert; Professor of Surgery in King's College, London, and Surgeon to King's College Hospital; Consulting Surgeon to the Westminster Hospital; 16, George-street, Hanover-sq. C. 1849-50.
- 1852 *ALFRED GEORGE FIELD, 28, Old Steyne, Brighton.
- 1850 *FREDERICK FIELD, Birmingham.
- 1849 GEORGE TUPMAN FINCHAM, M.D., Physician to the Westminster Hospital; 28, Chapel-street, Belgrave-square.
- 1836 JOHN WILLIAM FISHER, Surgeon-in-Chief to the Metropolitan Police Force; 5, Grosvenor-gate. C. 1843-4.
- 1838 GEORGE LIONEL FITZMAURICE, 97, Gloucester-place, Portman-square.
- 1842 THOMAS BELL ELCOCK FLETCHER, M.D., Physician to the General Hospital, Birmingham.
- 1848 JOHN GREGORY FORBES, 9, Devonport-street, Hyde-park.
- 1852 JOHN COOPER FORSTER, Assistant-Surgeon to Guy's Hospital; 11, Wellington-street, Southwark.
- 1817 *ROBERT THOMAS FORSTER, Southwell, Notts.
- 1820 THOMAS FORSTER, M.D., Hartfield Lodge, East Grinstead.
- 1856 JOHN F. FRANCE, Surgeon to the Eye Infirmary, and Lecturer on Ophthalmic Surgery at Guy's Hospital; 24, Bloomsbury-square.

Elected

- 1816 JOHN W. FRANCIS, M.D., Professor of Materia Medica in the University of New York, U.S.
- 1841 JOHN CHRISTOPHER AUGUSTUS FRANZ, M.D., Royal German Spa, Brighton.
- 1843 PATRICK FRASER, M.D., Physician to the London Hospital ; 46, Guilford-street, Russell-square.
- 1836 JOHN GEORGE FRENCH, Surgeon to the St. James's Infirmary ; 41, Great Marlborough-street. C. 1852-3.
- 1849 ROBERT TEMPLE FRERE, M.D., Physician-Accoucheur to, and Lecturer on Midwifery at, the Middlesex Hospital ; 9, Queen-street, May-fair.
- 1846 HENRY WILLIAM FULLER, M.D., Assistant-Physician to, and Lecturer on Medical Jurisprudence at, St. George's Hospital ; 13, Manchester-square.
- 1815 *GEORGE FREDERICK FURNIVAL, Egham, Surrey.
- 1854 ALFRED BARING GABROD, M.D., Professor of Materia Medica, Therapeutics, and Clinical Medicine in University College, London, and Physician to University College Hospital ; 84, Harley-street, Cavendish-square.
- 1851 GEORGE GASKOIN, 3, Westbourne-park.
- 1819 JOHN SAMUEL GASKOIN, 32, Clarges-street, Piccadilly. C. 1836.
- 1819 HENRY GAULTER.
- 1848 JOHN GAY, 10, Finsbury-place-south.
- 1821 *RICHARD FRANCIS GEORGE, Surgeon to the Bath Hospital.
- 1854 BERNARD GILPIN, Belle Vue House, Ulverstone, Lancashire.
- 1851 STEPHEN JENNINGS GOODFELLOW, M.D., Assistant-Physician to, and Lecturer on Medicine at, the Middlesex Hospital ; 4, Russell-square.
- 1818 JAMES ALEXANDER GORDON, M.D. C.B. F.R.S., Burford-lodge, Box-hill. C. 1828. V.P. 1829.
- 1851 PETER YEAMES GOWLLAND, Demonstrator of Anatomy at the London Hospital Medical College ; 34, Finsbury-square.
- 1844 JOHN GRANTHAM, Crayford, Kent.
- 1850 HENRY GRAY, F.R.S., Surgeon to the St. George's and St. James's Dispensary ; Lecturer on Anatomy at St. George's Hospital ; 8, Wilton-street, Grosvenor-place.

Elected

- 1846 GEORGE THOMPSON GREAM, M.D., 2, Upper Brook-street, Grosvenor-square.
- 1816 JOSEPH HENRY GREEN, F.R.S., Vice-President of the Royal College of Surgeons; Consulting Surgeon to St. Thomas's Hospital, Hadley, Middlesex. C. 1820. V.P. 1830.
- 1843 ROBERT GREENHALGH, M.D., 11, Upper Woburn-place, Russell-square.
- 1814 JOHN GROVE, M.D., Salisbury.
- 1852 JOHN GROVE, Wandsworth, Surrey.
- 1849 WILLIAM WITHEY GULL, M.D., Assistant-Physician to, and Lecturer on Physiology and Pathology at, Guy's Hospital; 8, Finsbury-square.
- 1837 JAMES MANBY GULLY, M.D., Holyrood House, Great Malvern.
- 1819 JOHN GUNNING, C.B., Inspector-General of Hospitals; Paris. C. 1823.
- 1842 CHARLES WILLIAM GARDINER GUTHRIE, Surgeon to, and Lecturer on Surgery at, the Westminster Hospital, and Surgeon to the Westminster Ophthalmic Hospital; 18, Pall Mall East.
- 1854 SAMUEL OSBORNE HABERSHON, M.D., Assistant-Physician to, and Lecturer on Materia Medica and Therapeutics at, Guy's Hospital; 48, Finsbury-circus.
- 1849 HAMMETT HAILEY, Newport Pagnell, Bucks.
- 1852 ROBERT JAMES HALE, M.D.
- 1845 JOHN HALL, M.D. K.C.B., Inspector-General of Hospitals.
- 1848 ALEXANDER HALLEY, M.D., F.G.S., 7, Harley-street, Cavendish-square.
- 1819 THOMAS HAMMERTON, 111, Piccadilly. C. 1829-30.
- 1838 HENRY HANCOCK, Surgeon to the Charing Cross Hospital, and to the Westminster Ophthalmic Hospital; 37, Harley-street, Cavendish-square. C. 1851.
- 1849 *RICHARD JAMES HANSARD, Oxford.
- 1848 *GEORGE HARCOURT, M.D., Chertsey, Surrey.
- 1836 JOHN FOSSE HARDING, 6, Mylne-street, Myddleton-square.
- 1843 THOMAS SUNDERLAND HARRISON, M.D. F.L.S., Garston Lodge, Somersetshire.
- 1846 JOHN HARRISON, 2, the Court-yard, Albany.

Elected

- 1841 WILLIAM HARVEY, Surgeon to the Royal Dispensary for Diseases of the Ear, and to the Freemasons' Female Charity; 2, Soho-square. C. 1854.
- 1853 ARTHUR HILL HASSALL, M.D., F.L.S., Physician to the Royal Free Hospital; 8, Bennett-street, St. James's.
- 1855 ALFRED HAVILAND, Bridgewater, Somerset.
- 1828 CÉSAR HENRY HAWKINS, F.R.S., *President*; Senior Surgeon to St. George's Hospital; 26, Grosvenor-street. C. 1830-1. V.P. 1838-9. T. 1841-4. P. 1855-6.
- 1838 CHARLES HAWKINS, Consulting Surgeon to Queen Charlotte's Hospital; 22, Savile-row, Burlington-gardens. C. 1846-7. S. 1850.
- 1848 THOMAS HAWKSLEY, M.D., Physician to the Dispensary for Consumption, Margaret-street; 26, George-street, Hanover-square.
- 1820 THOMAS EMERSON HEADLAM, M.D., Newcastle-upon-Tyne.
- 1848 *JAMES NEWTON HEALE, M.D., Physician to the Winchester County Hospital; Winchester.
- 1850 GEORGE HEATON, M.D., Boston, U.S.
- 1829 THOMAS HEBERDEN, M.D., 72, Park-street, Grosvenor-square.
- 1844 JOHN HENNEN, M.D. L. 1848-50.
- 1849 AMOS HENRIQUES, M.D., 67, Upper Berkeley-street, Portman-square.
- 1848 MITCHELL HENRY, Assistant-Surgeon to, and Lecturer on Medical Jurisprudence at, the Middlesex Hospital; 5, Harley-street, Cavendish-square.
- 1821 VINCENT HERBERSKI, M.D., Professor of Medicine in the University of Wilna.
- 1843 PRESCOTT GARDNER HEWETT, Professor of Anatomy and Surgery to the Royal College of Surgeons; Assistant-Surgeon to St. George's Hospital; 35, Hertford-street, May-fair.
- 1855 W. M. GRAILY HEWITT, M.D., Assistant-Physician to the Samaritan Free Hospital for Women and Children; Lecturer on Comparative Anatomy and Zoology at St. Mary's Hospital; 17, Radnor-place, Hyde-park.
- 1853 THOMAS HEWLETT, Surgeon to Harrow School; Harrow.
- 1841 *NATHANIEL HIGHMORE, Consulting-Surgeon to the Weymouth and Dorsetshire Eye Infirmary; Sherborne, Dorsetshire.

Elected

- 1814 *WILLIAM HILL, Wootton-under-Edge, Gloucestershire.
- 1854 THOMAS HILLIER, M.D. (Lond.), Medical Officer of Health to the Parish of St. Pancras; 21, Upper Gower-street.
- 1842 WILLIAM AUGUSTUS HILLMAN, Assistant-Surgeon to, and Lecturer on Anatomy and Physiology at, the Westminster Hospital; 1, Argyll-street, Regent-street.
- 1841 JOHN HILTON, F.R.S., Surgeon to, and Lecturer on Surgery at, Guy's Hospital; 10, New Broad-street, City. C. 1851.
- 1848 MARTIN THOMAS HISCOX, M.D., Bath, Somersetshire.
- 1840 THOMAS HODGKIN, M.D., Consulting Physician to the Hospital for Diseases of the Skin; 35, Bedford-square. C. 1842-3.
- 1813 JOSEPH HODGSON, F.R.S., 60, Westbourne-terrace, Hyde Park-gardens. C. 1817. P. 1851-2.
- 1835 THOMAS HENRY HOLBERTON, Hampton, Middlesex.
- 1843 LUTHER HOLDEN, Demonstrator of Anatomy at St. Barthomew's Hospital; 54, Gower-street, Bedford-square.
- 1814 SIR HENRY HOLLAND, Bart., M.D. F.R.S., Physician to the Queen, and Physician in Ordinary to H.R.H. Prince Albert; 25, Brook-street, Grosvenor-square. C. 1817, 1833-4. V.P. 1826, 1840.
- 1846 BARNARD WIGHT HOLT, Surgeon to the Westminster Hospital; 5, Parliament-street.
- 1846 CARSTEN H. HOLTHOUSE, Surgeon to the Public Dispensary, Lincoln's Inn; Assistant-Surgeon to, and Lecturer on Anatomy at, the Westminster Hospital; 2, Storey's-gate.
- 1853 WILLIAM CHARLES HOOD, M.D., Medical Superintendent, Bethlem Hospital.
- 1819 *JOHN HOWELL, M.D. F.R.S.E., Deputy-Inspector-General of Military Hospitals; Honorary and Consulting Physician to the Bristol Royal Infirmary; Datchet, near Windsor.
- 1828 *EDWARD HOWELL, M.D., Swansea, Glamorganshire.
- 1844 EDWIN HUMBY, 1, Windsor-terrace, Maida-hill.
- 1822 ROBERT HUME, M.D. C.B., Inspector of Hospitals; Commissioner in Lunacy; 9, Curzon-street, May-fair. V.P. 1836.

Elected

- 1855 GEORGE MURRAY HUMPHREY, Surgeon to Addenbrooke's Hospital, Cambridge.
- 1840 HENRY HUNT, M.D., 68, Brook-street, Hanover-square.
- 1842 CHRISTOPHER HUNTER, Downham, Norfolk.
- 1849 EDWARD LAW HUSSEY, Surgeon to the Radcliffe Infirmary, Oxford.
- 1820 WILLIAM HUTCHINSON, M.D.
- 1840 CHARLES HUTTON, M.D., Physician to the Royal Infirmary for Children; Assistant-Physician to the General Lying-in Hospital; 26, Lowndes-street, Belgrave-square.
- 1848 GEORGE COCKBURN HYDE, 16, South Parade, Brompton.
- 1847 WILLIAM EDMUND IMAGE, Surgeon to the Suffolk General Hospital; Bury St. Edmund's, Suffolk.
- 1826 WILLIAM INGRAM, Midhurst, Sussex.
- 1845 *HENRY JACKSON, Surgeon to the Sheffield General Infirmary; St. James's-row, Sheffield.
- 1841 PAUL JACKSON, 26, Wimpole-street, Cavendish-square.
- 1847 THOMAS REYNOLDS JACKSON, 28, Charles-street, St. James's.
- 1841 MAXIMILIAN MORITZ JACBOVICZ, M.D., Pesth.
- 1825 JOHN B. JAMES, M.D.
- 1847 *WILLIAM WITHALL JAMES, Exeter, Devonshire.
- 1844 SAMUEL JOHN JEAFRESON, M.D., Leamington, Warwickshire.
- 1839 JULIUS JEFFREYS, F.R.S., Kingston, Surrey.
- 1840 *GEORGE SAMUEL JENKS, M.D., Physician to the Sussex County Hospital, Brighton.
- 1851 WILLIAM JENNER, M.D., Professor of Pathological Anatomy in University College, London, and Physician to University College Hospital; and Physician to the Hospital for Sick Children; 8, Harley-street, Cavendish-square.
- 1848 ATHOL ARCHIBALD WOOD JOHNSON, Lecturer on Physiology at St. George's Hospital Medical School, and Surgeon to the Hospital for Sick Children; 37, Albemarle-street.
- 1851 EDMUND CHARLES JOHNSON, M.D., 6, Savile-row, and 20, Arlington-street.
- 1821 SIR EDWARD JOHNSON, M.D., Weymouth, Dorsetshire.
- 1847 GEORGE JOHNSON, M.D., Physician to King's College Hospital; 3, Woburn-square.

Elected

- 1837 HENRY CHARLES JOHNSON, Surgeon to St. George's Hospital; 6, Savile-row, Burlington-gardens. C. 1850-1.
- 1844 JOHN JOHNSTON.
- 1853 HENRY JONES, 23, Soho-square.
- 1844 HENRY BENICE JONES, M.D., F.R.S., Physician to St. George's Hospital; 31, Brook-street, Grosvenor-square. C. 1855-6.
- 1835 HENRY DERVICHE JONES, 23, Soho-square. C. 1854-5.
- 1853 THOMAS WHARTON JONES, F.R.S., Ophthalmic Surgeon to University College Hospital, and Professor of Ophthalmic Surgery in University College; 35, George-street, Hanover-square.
- 1837 THOMAS WILLIAM JONES, M.D., Physician to the City Dispensary; 19, Finsbury-pavement.
- 1829 *GEORGE CHARLES JULIUS, Richmond, Surrey.
- 1816 *GEORGE HERMANN KAUFFMANN, M.D., Hanover.
- 1815 ROBERT KEATE, Serjeant-Surgeon to the Queen, and Surgeon to H.R.H. the Duchess of Gloucester; 11, Hertford-street, May-fair. C. 1818-9. V.P. 1826, 1844.
- 1848 *DANIEL BURTON KENDELL, M.D., St. John's, Wakefield, Yorkshire.
- 1847 ALFRED KEYSER, 21, Norfolk-crescent, Oxford-square.
- 1839 *DAVID KING, M.D., Eltham, Kent.
- 1851 JOHN ABERNETHY KINGDON, Surgeon to the City Dispensary; 2, New Bank-buildings, City.
- 1840 SAMUEL ARMSTRONG LANE, Surgeon to the Lock Hospital; Surgeon to, and Lecturer on Anatomy at, St. Mary's Hospital; 1, Grosvenor-place. C. 1849-50.
- 1855 JAMES ROBERT LANE, Assistant-Surgeon to, and Lecturer on Anatomy and Physiology at, St. Mary's and the Lock Hospitals; 1, Grosvenor-place.
- 1841 *CHARLES LASHMAR, M.D., Croydon, Surrey.
- 1816 G. E. LAWRENCE.
- 1809 WILLIAM LAWRENCE, F.R.S., Surgeon Extraordinary to the Queen; Surgeon to, and Lecturer on Surgery at, St. Bartholomew's Hospital, and Surgeon to Bridewell and Bethlem Hospital; 18, Whitehall-place. S. 1813-7. V.P. 1818-9. C. 1820. T. 1821-6. P. 1831-2. C. 1833-4, 1842-3.

Elected

- 1840 THOMAS LAYCOCK, M.D., Professor of the Practice of Medicine in the University of Edinburgh.
- 1843 *JESSE LEACH, Heywood, near Bury, Lancashire.
- 1823 JOHN G. LEATH, M.D.
- 1822 JOHN JOSEPH LEDSAM, M.D.
- 1822 ROBERT LEE, M.D. F.R.S., Physician to the British Lying-in Hospital; Physician-Accoucheur to the St. Mary-lebone Infirmary; and Obstetric Physician to, and Lecturer on Midwifery at, St. George's Hospital; 4, Savile-row, Burlington-gardens. C. 1829, 1834. S. 1830-3. V.P. 1835.
- 1823 HENRY LEE, M.D. F.L.S., Weather Oak, Alvechurch, near Bromsgrove. C. 1837. S. 1839-40.
- 1843 HENRY LEE, Surgeon to King's College and the Lock Hospitals; 13, Dover-street, Piccadilly. C. 1856.
- 1851 GEORGE MACARTNEY LEESE, 50, Gloucester-place, Portman-square.
- 1836 FREDERICK LEIGHTON, M.D., Frankfort-on-the-Maine.
- 1854 HANANEL DE LEON, M.D., 2, Hampden-villas, Sandgate-road, Folkestone.
- 1856 DAVID LEWIS, M.D., Physician to the Royal General Dispensary, and to the Royal Society of Ancient Britons' Schools; 23, Finsbury-place.
- 1847 SIR JOHN LIDDELL, M.D. F.R.S. C.B., Director-General of the Medical Department of the Navy; Somerset House.
- 1806 JOHN LIND, M.D.
- 1845 WILLIAM JOHN LITTLE, M.D., Physician to the London Hospital; 34, Brook-street, Grosvenor-square.
- 1819 ROBERT LLOYD, M.D.
- 1824 EUSEBIUS ARTHUR LLOYD, Surgeon to St. Bartholomew's and Christ's Hospitals; 14, Bedford-row. S. 1827-8. V.P. 1838. C. 1843-4.
- 1820 J. G. LOCHER, M.C.D., Town Physician of Zurich.
- 1824 CHARLES LOCOCK, M.D., First Physician-Accoucheur to the Queen, and Consulting Physician to the General Lying-in-Hospital; 26, Hertford-street, May-fair. C. 1826. V.P. 1841.
- 1852 CHARLES LODGE, M.D.
- 1846 HENRY THOMAS LOMAX, Stafford.

Elected

- 1844 EDWARD FRANCIS LONSDALE, Surgeon to the Royal Orthopædic Hospital; 26, Montague-street, Russell-square.
- 1836 JOSEPH S. LÖWENFELD, M.D., Berbice.
- 1815 *PETER LUARD, M.D.
- 1852 JAMES LUKE, F.R.S., Senior Surgeon to the London Hospital; Surgeon to St. Luke's Hospital; 37, Broad-street-buildings, City.
- 1846 WILLIAM M'EWEN, M.D., Surgeon to the Cheshire County Gaol, and House-Surgeon to the Chester General Infirmary; Newgate-street, Chester.
- 1814 SIR JAMES MACGRIGOR, Bart., M.D. K.C.B. K.T.S. LL.D. F.R.S., Director-General of the Medical Department of the Army; 3, Harley-street, Cavendish-square. C. 1820. V.P. 1815-16. C. 1834-5.
- 1823 GEORGE MACILWAIN, Consulting Surgeon to the Finsbury Dispensary; 3, the Court-yard, Albany. C. 1829-30. V.P. 1848.
- 1848 FREDERICK WILLIAM MACKENZIE, M.D., Physician to the Western General Dispensary; 11, Chester-place, Hyde-park-square.
- 1818 WILLIAM MACKENZIE, Surgeon to the Eye Infirmary, Glasgow.
- 1854 *DRAPER MACKINDER, M.D., Gainsborough, Lincolnshire.
- 1822 RICHARD MACINTOSH, M.D.
- 1844 DANIEL MACLACHLAN, M.D., Physician to the Royal Hospital, Chelsea, and Deputy-Inspector-General of Hospitals; Royal Hospital, Chelsea.
- 1851 SAMUEL MACLEAN, 11, Brook-street, Grosvenor-square.
- 1849 DUNCAN MACLACHLAN MACLURE, 14, Harley-street, Cavendish-square.
- 1842 JOHN MACNAUGHT, M.D., Bedford-street, Liverpool.
- 1835 DANIEL CHAMBERS MACREIGHT, M.D., St. Hillier's, Jersey.
- 1837 ANDREW MELVILLE M'WHINNIE, Assistant-Surgeon to, and Lecturer on Comparative Anatomy at, St. Bartholomew's Hospital; Assistant-Surgeon to the London Hospital for Diseases of the Skin; 5, Crescent, New Bridge-street, Blackfriars. C. 1851-2.
- 1855 WILLIAM MARCET, M.D., Assistant-Physician to, and Lecturer on Physiological Chemistry at, the Westminster Hospital; 36, Chapel-street, Belgrave-square.

Elected

- 1848 WILLIAM ORLANDO MARKHAM, M.D., Assistant-Physician to, and Lecturer on Pathological Anatomy at, St. Mary's Hospital; 33, Clarges-street, Piccadilly.
- 1824 SIR HENRY MARSH, Bart., M.D., Dublin.
- 1838 THOMAS PARR MARSH, M.D., Physician to the Salop Infirmary, Shrewsbury.
- 1851 JOHN MARSHALL, Assistant-Surgeon to University College Hospital; 10, Savile-row, Burlington-gardens.
- 1841 JAMES RANALD MARTIN, F.R.S., 71A, Grosvenor-street. C. 1853.
- 1849 GEORGE BELLASIS MASFEN, 78, Oxford-street, Manchester.
- 1853 WILLIAM EDWARD MASFEN, Stafford.
- 1818 J. P. MAUNOIR, Professor of Surgery at Geneva.
- 1837 THOMAS MAYO, M.D. F.R.S., Physician to the St. Marylebone Infirmary; 56, Wimpole-street, Cavendish-square. S. 1841. C. 1847-8. V.P. 1851-2.
- 1839 RICHARD HENRY MEADE, Bradford, Yorkshire.
- 1837 SAMUEL WILLIAM JOHN MERRIMAN, M.D., Physician to the Royal Infirmary for Women and Children; Consulting Physician to the Westminster General Dispensary; and Physician-Accoucheur to the Western General Dispensary; 3, Charles-street, Westbourne-terrace. C. 1851-2.
- 1852 JAMES MERRYWEATHER, 57, Brook-street, Grosvenor-square.
- 1847 EDWARD MERYON, M.D., 14, Clarges-street, Piccadilly.
- 1815 AUGUSTUS MEYER, M.D., St. Petersburg.
- 1840 RICHARD MIDDLEMORE, Consulting-Surgeon to the Eye Infirmary, Birmingham.
- 1854 EDWARD ARCHIBALD MIDDLESHP, Richmond, Surrey.
- 1818 *PATRICK MILLER, M.D. F.R.S. E., Physician to the Devon and Exeter Hospitals, and to the Lunatic Asylum; Exeter, Devonshire.
- 1848 GAVIN MILBOY, M.D., 55, Victoria-street, Westminster.
- 1852 JAMES MONRO, M.D., Surgeon-Major, Coldstream Guards; Vincent-square, Westminster.
- 1844 NATHANIEL MONTEFIORE, 36, Hyde-park-gardens.
- 1836 GEORGE MOORE, M.D., Hastings.

Elected

- 1848 CHARLES HEWITT MOORE, Surgeon to, and Lecturer on Anatomy at, the Middlesex Hospital; 35, Montague-place, Russell-square.
- 1854 GEORGE MOSELEY, Sandgate, Kent.
- 1851 FREDERICK JOHN MOUAT, M.D., Professor of Medicine in the Medical College of Calcutta, and Secretary of the Council of Education in India; Calcutta.
- 1814 *GEORGE FREDERICK MUHRY, M.D., Hanover.
- 1856 CHARLES MURCHISON, M.D., Assistant-Physician to King's College Hospital; 21, Upper Seymour-st., Portman-sq.
- 1847 SIMON MURCHISON, Bicester, Oxon.
- 1845 THOMAS D. MUTTER, M.D., Professor of Surgery in Jefferson Medical College; Philadelphia.
- 1840 ROBERT NAIRNE, M.D., Physician to, and Lecturer on Medicine at, St. George's Hospital; 44, Charles-street, Berkeley-square. C. 1848-9. T. 1852-3.
- 1835 THOMAS ANDREW NELSON, M.D., 10, Nottingham-terrace, York-gate, Regent's-park.
- 1843 EDWARD NEWTON, 30, Fitzroy-square.
- 1851 JAMES NICHOLS, 13, Savile-row, Burlington-gardens.
- 1819 *GEORGE NORMAN, Surgeon to the United Hospital and Puerperal Charity; Bath.
- 1849 HENRY BURFORD NORMAN, Consulting Surgeon to the North St. Pancras Provident Eye Infirmary, and to the St. Marylebone Charity Schools; 7, Manchester-square.
- 1845 HENRY NORRIS, South Petherton, Somerset.
- 1849 *ARTHUR NOVERRE, Great Stanmore, Middlesex.
- 1847 *WILLIAM EDWARD CHARLES NOURSE, Ivy House, West Cowes, Isle of Wight.
- 1843 WILLIAM O'CONNOR, M.D., Assistant-Physician to the Royal Free Hospital; 30, Upper Montagu-street, Montagu-sq.
- 1847 THOMAS O'CONNOR, March, Cambridgeshire.
- 1846 FRANCIS ODLING, 52, Devonshire-street, Portland-place.
- 1822 JAMES ADEY OGLE, M.D. F.R.S., Clinical and Aldrichian Professor of Medicine, Oxford; and Senior Physician to the Radcliffe Infirmary; Oxford.
- 1855 WILLIAM OGLE, M.D., Physician to the Royal Pimlico Dispensary; 9, Lower Belgrave-street, Belgrave-square.
- 1850 HENRY OLDHAM, M.D., Obstetric Physician to Guy's Hospital; 26, Finsbury-square.

Elected

- 1842 WILLIAM PIERES ORMEROD.
- 1846 *EDWARD LATHAM ORMEROD, M.D., Physician to the Sussex County Hospital; Old Steyne, Brighton.
- 1847 WILLIAM EMANUEL PAGE, M.D., Physician to, and Lecturer on Medicine at, St. George's Hospital; 11, Queen-street, May-fair.
- 1847 *WILLIAM BOUSFIELD PAGE, Surgeon to the Cumberland Infirmary; Carlisle.
- 1840 JAMES PAGET, F.R.S., Assistant-Surgeon to, and Lecturer on General and Morbid Anatomy and Physiology at, St. Bartholomew's Hospital; 24, Henrietta-street, Cavendish-square. C. 1848-9.
- 1806 *ROBERT PALEY, M.D., Bishopston Grange, near Ripon, Yorkshire.
- 1836 S. W. LANGSTON PARKER, Surgeon to the Queen's Hospital; Birmingham.
- 1847 NICHOLAS PARKER, M.D., Assistant-Physician to, and Lecturer on Medicine at, the London Hospital; 22, Finsbury-square.
- 1841 JOHN PARKIN, M.D., Paris.
- 1851 JAMES PART, 7, Camden-road-villas, Camden-town.
- 1828 RICHARD PARTRIDGE, F.R.S., Surgeon to King's College Hospital, and Professor of Anatomy in King's College, London; 17, New-street, Spring-gardens. S. 1832-6. C. 1837-8. V.P. 1847-8.
- 1845 THOMAS BEVILL PEACOCK, M.D., *Secretary*; Assistant-Physician to, and Lecturer on Materia Medica at, St. Thomas's Hospital; Physician to the City of London Hospital for Diseases of the Chest, Victoria Park; 20, Finsbury-circus. S. 1855.
- 1856 RICHARD KING PEIRCE, 16, Norland-place, Notting-hill.
- 1830 CHARLES P. PELECHIN, M.D., St. Petersburg.
- 1855 *OLIVER PEMBERTON, Surgeon to the General Hospital at Birmingham, and Demonstrator of Anatomy at Queen's College; 11, Temple-row, Birmingham.
- 1844 WILLIAM VESALIUS PETTIGREW, M.D., 7, Chester-street, Grosvenor-place.
- 1837 BENJAMIN PHILLIPS, F.R.S., Brentbridge House, Hendon, Middlesex. L. 1841-5. T. 1847-50. V.P. 1853.

Elected

- 1814 *EDWARD PHILLIPS, M.D., Consulting Physician to the County Hospital; Winchester, Hampshire.
- 1848 EDWARD PHILLIPS, M.D., Coventry, Warwickshire.
- 1852 RICHARD PHILLIPS, 5, Winchester-place, Pentonville.
- 1854 THOMAS BACON PHILLIPS, 36, Lansdown-place, Brighton.
- 1846 FRANCIS RICHARD PHILP, M.D.
- 1851 *JAMES HOLLINS PICKFORD, M.D. M.R.I.A., Brighton.
- 1851 JOHN PICTON, M.D.
- 1836 ISAAC PIDDUCK, M.D., Physician to the Bloomsbury Dispensary; 22, Montague-street, Russell-square.
- 1852 HENRY PILLEAU, Staff Surgeon; 21, Kensington-square.
- 1841 HENRY ALFRED PITMAN, M.D., Assistant-Physician to, and Lecturer on Materia Medica at, St. George's Hospital; 28, Montague-place, Russell-square. L. 1851-3.
- 1850 ALFRED POLAND, Assistant-Surgeon to Guy's Hospital, and Surgeon to the Royal Ophthalmic Hospital, Moorfields; 4, St. Thomas's-street, Southwark.
- 1845 GEORGE DAVID POLLOCK, Surgeon to the North London Eye Infirmary; Assistant-Surgeon to, and Lecturer on Anatomy at, St. George's Hospital; 27, Grosvenor-street. C. 1856.
- 1843 CHARLES POPE, M.D. F.L.S., Temple Cloud, near Bristol.
- 1846 JEPHSON POTTER, M.D. F.L.S., Oxford-road, Manchester.
- 1842 JAMES POWELL, M.D. (Lond.), 77, Guildford-street, Russell-square.
- 1840 LEWIS POWELL, 13, John-street, Berkeley-square.
- 1851 ROBERT FRANCIS POWER, M.D., 14, Waterloo-place.
- 1839 JOHN PROPERT, 6, New Cavendish-street, Portland-place.
- 1845 JOHN PYLE, Surgeon to the North London Eye Infirmary; 56, Oxford-terrace, Hyde-park.
- 1816 SIR WILLIAM PYM, M.D., Inspector of Hospitals.
- 1830 JONES QUAIN, M.D., Paris.
- 1850 RICHARD QUAIN, M.D., Physician to the Hospital for Consumption, and Diseases of the Chest; 23, Harley-street, Cavendish-square.
- 1835 RICHARD QUAIN, F.R.S., *Vice-President*, Surgeon to University Hospital; Consulting Surgeon to the Eye Infirmary; and Professor of Clinical Surgery in University College, London; 32, Cavendish-square. C. 1838-9. L. 1846-8. T. 1851-3.

Elected

- 1852 CHARLES BLAND RADCLIFFE, M.D., Assistant-Physician to, and Lecturer on Materia Medica at, the Westminster Hospital; 4, Henrietta-street, Cavendish-square.
- 1854 WILLIAM HENRY RANSOM, M.D., Nottingham.
- 1821 HENRY REEDER, M.D., Ridge House, Chipping, Sudbury.
- 1835 G. REGNOLI, Professor of Surgery in the University of Pisa.
- 1855 JOHN RUSSELL REYNOLDS, M.D., Assistant-Physician to the Hospital for Sick Children; 38, Grosvenor-street.
- 1847 SAMUEL RICHARDS, M.D., 36, Bedford-square.
- 1852 CHRISTOPHER THOMAS RICHARDSON, M.B., Physician to the Metropolitan Free Hospital; 16, Hinde-street, Manchester square.
- 1829 SIR JOHN RICHARDSON, Knt., F.R.S. C.B.
- 1849 *WILLIAM RICHARDSON, M.D., 9, Ephraim-road, Tunbridge Wells, Kent.
- 1845 BENJAMIN RIDGE, M.D., Putney, Surrey.
- 1843 JOSEPH RIDGE, M.D., 39, Dorset-square.
- 1852 CHARLES RIDLEY, 6, Charlotte-street, Bedford-square.
- 1852 JOHN ROBERTS, M.D., 75, Grosvenor-street.
- 1829 *ARCHIBALD ROBERTSON, M.D. F.R.S., Northampton.
- 1855 CHARLES ALEXANDER LOCKHART ROBERTSON, M.D., Hon. Secretary to the Association of Medical Officers of Asylums and Hospitals for the Insane; 1, Charles-street, Berkeley-square.
- 1843 GEORGE ROBINSON, M.D., Newcastle-on-Tyne.
- 1843 WILLIAM RODEN, M.D. F.L.S., Kidderminster.
- 1835 GEORGE HAMILTON ROE, M.D., Senior Physician to the Westminster Hospital and to the Hospital for Consumption and Diseases of the Chest; 56, Park-street, Grosvenor-square. C. 1841-2.
- 1836 ARNOLD ROGERS, 16, Hanover-square.
- 1846 WILLIAM RICHARD ROGERS, M.D., 56, Berners-street.
- 1819 HENRY SHUCKBURGH ROOTS, M.D., Consulting Physician to St. Thomas's Hospital; 2, Russell-square. C. 1833. V.P. 1834-5. C. 1845.
- 1829 WILLIAM SUDLOW ROOTS, Kingston, Surrey.
- 1850 GEORGE ROPER, 180, Shoreditch.
- 1836 RICHARD ROSCOE, M.D., Twickenham, Middlesex.
- 1855 THOMAS TATTERSALL ROSCOW, M.D., 1, Sumner-pl., Brompton.
- 1836 *CALEB BURRELL ROSE, Swaffham, Norfolk.

Elected

- 1849 CHARLES HENRY FELIX ROUTH, M.D., 52, Montagu-square.
 1845 HENRY MORTIMER ROWDON, 29, Nottingham-place, York-gate, Regent's-park.
 1834 HENRY WILLIAM RUMSEY, Cheltenham.
 1845 JAMES RUSSELL, M.D., Physician to the General Dispensary, Birmingham.
 1851 HENRY HYDE SALTER, M.D. F.R.S., Assistant-Physician to, and Lecturer on Physiology and Pathology at, the Charing Cross Hospital; 6, Montague-street, Russell-sq.
 1856 SAMUEL JAMES A. SALTER, 17, New Broad-street, City.
 1844 *THOMAS BELL SALTER, M.D. F.L.S., Ryde, Isle of Wight.
 1849 HUGH JAMES SANDERSON, M.D., 26, Upper Berkeley-street, Portman-square.
 1855 JOHN BURDON SANDERSON, M.D., Medical Officer of Health for Paddington; Lecturer on Medical Jurisprudence at St. Mary's Hospital; 9, Gloucester-place, Hyde-park.
 1847 WILLIAM HENRY OCTAVIUS SANKEY, M.D., Middlesex County Lunatic Asylum, Hanwell.
 1845 EDWIN SAUNDERS, Surgeon-Dentist to the Queen, 13A, George-street, Hanover-square.
 1834 LUDWIG V. SAUVAN, M.D., Warsaw.
 1840 AUGUSTIN SAYER, M.D., Physician to the Lock Hospital, 28, Upper Seymour-street, Portman-square.
 1853 MAURICE SCHULHOF, M.D., Physician to the Royal General Dispensary, Bartholomew-close; 7, Suffolk-place, Pall Mall.
 1856 EDWIN SERCOMBE, Surgeon-Dentist to the Paddington Free Dispensary; 6, Somers-place, Hyde-park.
 1824 EDWARD JAMES SEYMOUR, M.D. F.R.S., 13, Charles-street, Berkeley-square. C. 1826, 1831. S. 1827-8. V.P. 1830, 1842.
 1840 WILLIAM SHARP, F.R.S., Rugby.
 1837 WILLIAM SHARPEY, M.D. F.R.S. Professor of Anatomy and Physiology in University College, London, and Secretary of the Royal Society; 33, Woburn-place, Russell-square. C. 1848-9.
 1836 ALEXANDER SHAW, Surgeon to, and Lecturer on Surgery at, the Middlesex Hospital; 25, Henrietta-street, Cavendish-square. C. 1842. S. 1843-4. V.P. 1851-2.
 1848 *EDWARD JAMES SHEARMAN, M.D., Rotherham, Yorkshire.

Elected

- 1849 FRANCIS SIBSON, M.D. F.R.S., Physician to, and Lecturer on Medicine at, St. Mary's Hospital; 40, Brook-street, Grosvenor-square.
- 1848 EDWARD HENRY SIEVEKING, M.D., Assistant-Physician to, and Lecturer on Materia Medica at, St. Mary's Hospital; 3, Bentinck-street, Manchester-square.
- 1839 THOMAS HOOKHAM SILVESTER, M.D., High-street, Clapham. C. 1854-5.
- 1842 JOHN SIMON, F.R.S., Surgeon to, and Lecturer on Pathology at, St. Thomas's Hospital; Medical Officer of the General Board of Health; 1, Cumberland-street. C. 1854-5.
- 1821 CHARLES SKENE, M.D., Professor of Anatomy and Surgery; Marischal College, Aberdeen.
- 1827 GEORGE ROBERT SKENE, Bedford.
- 1824 FREDERIC CARPENTER SKEY, F.R.S., Surgeon to, and Lecturer on Anatomy at, St. Bartholomew's Hospital; 13, Grosvenor-street. C. 1828. L. 1829-31. V.P. 1841-2.
- 1852 CHARLES CASE SMITH, Senior Surgeon to the Suffolk General Hospital; Bury St. Edmunds, Suffolk.
- 1854 EDWARD SMITH, M.D. LL.B., Assistant-Physician to the Hospital for Consumption and Diseases of the Chest; 63, Grosvenor-street.
- 1835 JOHN GREGORY SMITH, Harewood, Yorkshire.
- 1843 ROBERT WILLIAM SMITH, M.D. M.R.I.A., Professor of Surgery in the University of Dublin; Surgeon to the Richmond Hospital; 63, Eccles-street, Dublin.
- 1838 SPENCER SMITH, *Secretary*; Senior Assistant-Surgeon to, and Lecturer on Surgery at, St. Mary's Hospital; 48, Sussex-gardens, Hyde-park. C. 1854. S. 1855.
- 1845 WILLIAM SMITH, Chesterfield, Derbyshire.
- 1847 WILLIAM SMITH, M.D., Weymouth, Dorsetshire.
- 1850 WILLIAM TYLER SMITH, M.D., Physician-Accoucheur to, Lecturer on Midwifery at, St. Mary's Hospital; 7, Upper Grosvenor-street.
- 1843 JOHN SNOW, M.D., 18, Sackville-street, Piccadilly.
- 1851 JOHN SODEN, Surgeon to the Bath Hospital; Surgeon to Bath United Hospital, and Bath Eye Infirmary; Bath.
- 1816 *JOHN SMITH SODEN, New Sidney-place, Bath.

Elected

- 1830 SAMUEL SOLLY, F.R.S., Surgeon to St. Thomas's Hospital ;
18, St. Helen's-place, Bishopsgate-street. L. 1838-40.
C. 1845-6. V.P. 1849-50.
- 1844 FREDERICK ROBERT SPACKMAN, M.B., Harpenden, St. Alban's.
- 1834 JAMES SPARK, Newcastle, Staffordshire.
- 1851 ROBERT JOHN SPITTA, M.B., Clapham, Surrey.
- 1843 *STEPHEN SPRANGER, 27, Henrietta-street, Bath.
- 1838 GEORGE JAMES SQUIBB, 11, Montagu-place, Montagu-square. C. 1856.
- 1815 EDWARD STANLEY, F.R.S., Vice-President of the Royal College of Surgeons ; Surgeon to St. Bartholomew's Hospital ; 23A, Brook-street, Grosvenor-square. C. 1821-2, 1835, 1845-6, 1852-3. S. 1824. V.P. 1827, 1839-40. T. 1832-4. P. 1843-4.
- 1851 JAMES STARTIN, Surgeon to the Hospital for Diseases of the Skin, and Lecturer on Cutaneous Disorders at that Institution ; 3, Savile-row, Burlington-gardens.
- 1852 SHEARD FREEMAN STATHAM, Surgeon to the Great Northern Hospital or Infirmary, King's Cross ; 43, Mortimer-street, Cavendish-square.
- 1854 HENRY STEVENS, Resident Medical Officer, St. Luke's Hospital.
- 1842 ALEXANDER PATRICK STEWART, M.D., Physician to, and Lecturer on Medicine at, the Middlesex Hospital ; 74, Grosvenor-street. C. 1856.
- 1856 ALONZO HENRY STOCKER, M.D., Resident Medical Superintendent of Grove Hall Asylum, Bow.
- 1843 ROBERT REEVE STORKS.
- 1839 ALEXANDER JOHN SUTHERLAND, M.D. F.R.S., Physician to St. Luke's Hospital ; 6, Richmond-terrace, Whitehall. C. 1850-1.
- 1855 JOHN MAULE SUTTON, M.D., Kent House, Tenby, South Wales.
- 1842 JAMES SYME, Professor of Clinical Surgery in the University of Edinburgh ; Charlotte-square, Edinburgh.
- 1854 *FREDERICK SYMONDS, Surgeon to the Radcliffe Infirmary ; 32, Beaumont-street, Oxford.
- 1844 RICHARD WILLIAM TAMPLIN, Surgeon to the Royal Orthopædic Hospital ; 33, Old Burlington-street.

Elected

- 1848 THOMAS HAWKES TANNER, M.D., Physician to the Hospital for Women, Soho-square; 10, Charlotte-street, Bedford-square.
- 1840 THOMAS TATUM, Surgeon to, and Lecturer on Surgery at, St. George's Hospital; 3, George-street, Hanover-square. C. 1852-3.
- 1835 JOHN COLLEY TAUNTON, Surgeon to the City of London Truss Society, and to the City Dispensary; 48, Hatton-garden, Holborn. C. 1840-1.
- 1852 ROBERT TAYLOR, Surgeon to the Central London Ophthalmic Hospital, and to the Cripple's Home, Hill-street; 10, George-street, Hanover-square.
- 1845 THOMAS TAYLOR, Lecturer on Chemistry at the Middlesex Hospital Medical School; 4, Vere-street, Cavendish-square.
- 1856 THOMAS PRIDGIN TEALE, F.L.S., Surgeon to the Leeds General Infirmary; 22, Albion-street, Leeds.
- 1845 *EVAN THOMAS, Manchester.
- 1839 SETH THOMPSON, M.D., Physician to the Middlesex Hospital; 16, Lower Berkeley-street, Portman-square. C. 1849. S. 1850-1.
- 1842 THEOPHILUS THOMPSON, M.D. F.R.S., Physician to the Hospital for Consumption and Diseases of the Chest; 3, Bedford-square. C. 1855-6.
- 1852 HENRY THOMPSON, Surgeon to the St. Marylebone Infirmary, and Assistant-Surgeon to University College Hospital; 16, Wimpole-street, Cavendish-square.
- 1835 FREDERICK HALE THOMSON, Consulting Surgeon to the Westminster Hospital, and to the West London Institution for Diseases of the Eye; 4, Clarges-street, Piccadilly.
- 1819 JOHN THOMSON, M.D. F.L.S., Physician to the Finsbury Dispensary; 18, Dalby-terrace, Islington. C. 1833. L. 1834-7. V.P. 1850-1.
- 1850 ROBERT DUNDAS THOMSON, M.D. F.R.S., Professor of Chemistry at St. Thomas's Hospital, and Medical Officer of Health for the Parish of St. Marylebone; 11, Marlborough-hill, St. John's-wood.
- 1836 JOHN THURNAM, M.D., Devizes, Wiltshire.

Elected

- 1848 EDWARD JOHN TILT, M.D., Physician to the Farringdon Dispensary; 11, York-street, Portman-square.
- 1834 ROBERT BENTLEY TODD, M.D. F.R.S., Physician to King's College Hospital; 26, Brook-street, Grosvenor-square. L. 1842-6. T. 1850-1. V.P. 1854.
- 1828 JAMES TORRIE, M.D., Aberdeen.
- 1843 JOSEPH TOYNBEE, F.R.S., Aural Surgeon to St. Mary's Hospital, Consulting Aural Surgeon to the Asylum for the Deaf and Dumb, and to the St. George's and St. James's General Dispensary, and Lecturer on Aural Surgery at St. Mary's Hospital Medical School; 18, Savile-row, Burlington-gardens.
- 1850 SAMUEL JOHN TRACY, Surgeon-Dentist to St. Bartholomew's and Christ's Hospitals; 28, Old Burlington-street.
- 1808 BENJAMIN TRAVERS, F.R.S., President of the Royal College of Surgeons, Surgeon Extraordinary to the Queen, Surgeon in Ordinary to H.R.H. Prince Albert; 54, Green-street, Grosvenor-square. C. 1810, 1819, 1822-3, 1842, 1847. V.P. 1817-8. P. 1827-8.
- 1854 BENJAMIN TRAVERS, Jun., 8, Dover-street, Piccadilly.
- 1841 MATTHEW TRUMAN, M.D., 40, Norland-square, Notting-hill.
- 1855 JAMES TULLOCH, M.D., 1, Pembridge-place, Westbourne-grove-west.
- 1835 JOHN CUSSON TURNER, M.D., South-terrace, Bexley Heath, Kent.
- 1845 THOMAS TURNER, Surgeon to the Royal Manchester Infirmary, and Lecturer on Anatomy; Mosley-street, Manchester.
- 1846 ALEXANDER URE, Surgeon to, and Lecturer on Clinical Surgery at, St. Mary's Hospital, and Consulting Surgeon to the Westminster General Dispensary; 18, Upper Seymour-street, Portman-square.
- 1819 BARNARD VAN OVEN, M.D., Consulting Surgeon to the Charity for Delivering Jewish Lying-in Women; 22, Manchester-square.
- 1806 BOYER VAUX, M.D.
- 1810 JAMES VOSE.
- 1828 BENEDETTO VULPES, M.D., Physician to the Hospital of Aversa, and to the Hospital of Incurables, Naples.

Elected

- 1854 EDWARD WADDINGTON, 3d Staffordshire Regiment; Newcastle-under-Lyme.
- 1841 ROBERT WADE, Surgeon to the Westminster General Dispensary; 68, Dean-street, Soho.
- 1823 WILLIAM WAGNER, M.D., Berlin.
- 1820 THOMAS WALKER, M.D., Physician to the Forces; Morro Velho, Brasil.
- 1852 WALTER HAYLE WALSHE, M.D., Professor of the Theory and Practice of Medicine in University College, London, and Physician to University College Hospital; Consulting Physician to the Hospital for Consumption; 40, Queen Anne-street, Cavendish-square.
- 1851 HENRY HAYNES WALTON, Surgeon to the Central London Ophthalmic Hospital, and Assistant-Surgeon to St. Mary's Hospital; 69, Brook-street, Hanover-square.
- 1852 DANIEL WANE, M.D., Obstetric Physician to the Blenheim-street Dispensary; 20, Grafton-street, Berkeley-square.
- 1846 NATHANIEL WARD, Assistant-Surgeon to, and Lecturer on Anatomy at, the London Hospital; 1, Broad-street-buildings, City.
- 1845 THOMAS OGIER WARD, M.D., 9, Leonard-place, Kensington.
- 1821 WILLIAM TILLEARD WARD, Duncannon House, Brighton.
- 1846 JAMES THOMAS WARE, Surgeon to the Finsbury Dispensary and to the Convalescent Institution; 51, Russell-square.
- 1811 JOHN WARE, Clifton, near Bristol.
- 1814 MARTIN WARE, 51, Russell-square. C. 1844-5. T. 1846. V.P. 1853.
- 1816 *CHARLES BRUCE WARNER, Cirencester, Gloucestershire.
- 1829 ELIAS TAYLOR WARRY, Wimborne, Dorsetshire.
- 1837 THOMAS WATSON, M.D., Consulting Physician to King's College Hospital; 16, Henrietta-street, Cavendish-square. C. 1840-1, 1852. V.P. 1845-6.
- 1847 *THOMAS WATSON, Holbeach, Lincolnshire.
- 1854 WILLIAM WEBB, M.D., Resident Medical Officer of the Stafford General Infirmary; Wirksworth, Derbyshire.
- 1840 WILLIAM WOODHAM WEBB, Gislegham, near Thwaite, Suffolk.
- 1842 FREDERICK WEBER, M.D., Physician to the St. George's and St. James's Dispensary; 44, Green-street, Park-lane.

Elected

- 1835 JOHN WEBSTER, M.D. F.R.S., *Vice-President*; Physician to the Scottish Hospital; Consulting Physician to the St. George's and St. James's Dispensary; 24, Brook-street, Grosvenor-square. C. 1843-4. V.P. 1855.
- 1844 WILLIAM WEGG, M.D., *Librarian*; Physician to the St. George's and St. James's Dispensary; 5, Maddox-street, Hanover-square. L. 1854-5.
- 1854 THOMAS SPENCER WELLS, Lecturer on Surgery at the Grosvenor-place School of Medicine; 3, Upper Grosvenor-street.
- 1816 SIR AUGUSTUS WEST, Knt., Deputy-Inspector of Hospitals to the Portuguese Forces; Paris.
- 1842 CHARLES WEST, M.D., Physician-Accoucheur to, and Lecturer on Midwifery at, St. Bartholomew's Hospital; and Physician to the Hospital for Sick Children; 96, Wimpole-street, Cavendish-square. C. 1855-6.
- 1841 THOMAS WEST, M.D. F.L.S., Daventry.
- 1828 JOHN WHATLEY, M.D.
- 1849 JOHN WHITE.
- 1852 JOHN WIBLIN, 73, Morland-place, Southampton.
- 1840 JOSEPH WICKENDEN, Birmingham.
- 1824 *WILLIAM JOHN WICKHAM, Surgeon to the Winchester Hospital; Winchester.
- 1844 FREDERICK WILDBORE, 1, Trafalgar-place-east, Hackney-road.
- 1837 GEORGE AUGUSTUS FREDERICK WILKS, M.D., Temple-walk, Matlock, Derbyshire.
- 1840 CHARLES JAMES BLASIUS WILLIAMS, M.D. F.R.S., Consulting Physician to the Hospital for Consumption; 49, Upper Brook-street, Grosvenor-square. C. 1849-50.
- 1829 ROBERT WILLIS, M.D., Barnes, Surrey. L. 1838-41.
- 1839 ERASMUS WILSON, F.R.S., Consulting Surgeon to the St. Pancras Infirmary; 17, Henrietta-street, Cavendish-square.
- 1839 JAMES ARTHUR WILSON, M.D., Physician to St. George's Hospital; 28, Dover-street, Piccadilly. C. 1846-7.
- 1850 *ROBERT STANTON WISE, M.D., Banbury, Oxon.
- 1825 THOMAS ALEXANDER WISE, India.

Elected

- 1851 JOHN WOOD, Assistant-Surgeon to King's College Hospital ;
King's College, Strand.
- 1841 GEORGE LEIGHTON WOOD, Surgeon to the Bath Hospital ;
Queen-square, Bath.
- 1848 WILLIAM WOOD, M.D., 54, Upper Harley-street.
- 1843 JOHN WARD WOODFALL, M.D., Physician to the West Kent
Infirmary ; Maidstone, Kent.
- 1833 THOMAS WORMALD, Assistant-Surgeon to St. Bartholomew's
Hospital ; Surgeon to the Foundling Hospital ; 42, Bed-
ford-row. C. 1839. V.P. 1854.
- 1842 WILLIAM COLLINS WORTHINGTON, Surgeon to the Infir-
mary, Lowestoft, Suffolk.
- 1848 EDWARD JOHN WRIGHT, 13, Montague-place, Clapham-road.
- 1855 HENRY G. WRIGHT, M.D., Physician to the St. Pancras
Royal Dispensary ; 23, Somerset-street, Portman-square.

[It is particularly requested, that any change of Title or Residence may be communicated to the Secretaries before the 1st of August in each year, in order that the List may be made as correct as possible.]

HONORARY FELLOWS.

(Limited to Twelve.)

Elected

- 1841 WILLIAM THOMAS BRANDE, F.R.S., Professor of Chemistry at the Royal Institution of Great Britain; Royal Mint, Tower-hill.
- 1835 SIR DAVID BREWSTER, K.H. LL.D. F.R.S., &c., Corresp. Memb. Institute of France, &c.; St. Andrew's.
- 1853 BENJAMIN COLLINS BRODIE, B.A., Oxford, F.R.S., Professor of Chemistry in the University of Oxford.
- 1841 ROBERT BROWN, D.C.L. F.R.S., Keeper of Botany, British Museum.
- 1847 EDWIN CHADWICK, late Commissioner of the Board of Health.
- 1835 MICHAEL FARADAY, D.C.L. F.R.S., Corresp. Memb. Institute of France; Royal Institution.
- 1841 SIR JOHN FREDERICK WILLIAM HERSCHEL, Bart., D.C.L. F.R.S., Corresp. Memb. Institute of France; Collingwood, Kent.
- 1835 SIR WILLIAM JACKSON HOOKER, LL.D. F.R.S.; Royal Botanic Garden, Kew.
- 1847 RICHARD OWEN, F.R.S., Corresp. Memb. Institute of France; Superintendent of the Natural History Department in the British Museum.
- 1835 The Rev. ADAM SEDGWICK, A.M. F.R.S., &c., Woodwardian Lecturer, Cambridge.
- 1841 The Rev. WILLIAM WHEWELL, D.D. F.R.S., Master of Trinity College, Cambridge.

FOREIGN HONORARY FELLOWS.

(Limited to Twenty.)

Elected

- 1841 G. ANDRAL, M.D., Professor in the Faculty of Medicine, Paris.
- 1835 CARL JOHAN ECKSTRÖM, K.P.S. and W., Physician to the King of Sweden, First Surgeon to the Seraphim Hospital, Stockholm.
- 1841 CHRISTIAN GOTTFRIED EHRENBURG, Berlin.
- 1835 BARON ALEXANDER VON HUMBOLDT, Member of the Institute of France, &c., Berlin.
- 1841 JAMES JACKSON, M.D., Professor of Medicine in the University of Cambridge, Boston, U.S.
- 1843 BARON JUSTUS VON LIEBIG, M.D. F.R.S., Professor of Chemistry in the University of Munich, &c.
- 1841 P. C. A. LOUIS, M.D., Physician to the Hôtel-Dieu, Member of the Royal Academy of Medicine, &c., Paris.
- 1847 CARLO MATTEUCCI, Professor in the University of Pisa.
- 1853 VALENTINE MOTT, M.D., New York.
- 1841 JOHANNES MÜLLER, M.D., Professor of Anatomy and Physiology, and Director of the Royal Anatomical Museum, Berlin.
- 1841 BARTOLOMEO PANIZZA, M.D., Pavia.
- 1850 CARL ROKITANSKY, M.D., Curator of the Imperial Pathological Museum at the University of Vienna, &c &c.
- 1835 C. J. TEMMINCK, Director of the Museum of Natural History of the King of Holland, Amsterdam.
- 1835 FRIEDRICH TIEDEMANN, M.D., Frankfort-on-the-Maine.

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ON
THE ACTION OF DIGITALIS
UPON
THE UTERUS.

BY
W. HOWSHIP DICKINSON,
LATE OBSTETRIC ASSISTANT AT ST. GEORGE'S HOSPITAL.

COMMUNICATED BY
H. BENCE JONES, M.D. F.R.S

Received Nov. 3d, 1855—Read Nov. 27th, 1855.

LAST October, in the Burton Ward of St. George's, a case of most severe menorrhagia, which had nearly proved fatal, was cured by the infusion of digitalis, administered almost by accident. The circumstances are sufficiently striking to deserve a full account of the case.

Catharine Mackenzie, æt. 48, a widow, was admitted on the 18th of September, 1854. She stated that her health had been good, and that the uterine functions had been performed naturally, until twelve months previously, when she fell from a ladder, about twelve feet high, and got considerably bruised about the lower part of the back. A week after this blood began to flow from the vagina; at first in small quantities, but it soon became profuse and accom-

panied with pain ; and so it continued, without intermitting for a single day, until her admission.

She was first treated in another department of the hospital without benefit ; and when she was transferred to Dr. Lee, her case was regarded in the most unfavorable light.

She was perfectly blanched, and was scarcely able to stand, on account of weakness and vertigo. There was a discharge of blood from the vagina, so copious that none of the ordinary appliances were sufficient to keep it within bounds ; it poured into the bed ; and during micturition it passed from the vagina in gushes. There was slight pain about the sacrum. The ankles were œdematous ; the tongue flabby and rather coated ; the pulse 80, not very unnatural. On examination, the uterus was found in a perfectly healthy condition, though slightly anteverted ; the os was quite soft and smooth, and there was no organic disease of any kind within reach.

Nothing was made out which could give any particular direction to the treatment. Sulphuric acid and sulphate of magnesia were given, but with no effect, except as an aperient.

Three days after this the patient complained of pain in the region of the heart ; and said that during the night she had had several attacks of shortness of breath, in which she thought she should have died. On examining the chest, the signs of pericarditis were at once perceived, and the patient was referred to Dr. Nairne.

On the 28th of September all acute symptoms had subsided, and the only unnatural sound in the chest was a regurgitant aortic murmur. The hemorrhage was more profuse than ever, and the patient was extremely low. Four grains of acetate of lead, with half a grain of opium, were given three times a day, and wine was ordered.

On the 2d of October a violent attack of purging and vomiting, with much coldness of surface, made a change of treatment necessary. The discharge was not in the least diminished. On the following day medicine was given con-

taining sulphuric acid and chloric ether. This seemed to do rather harm than good; and, on the 5th, the acetate of lead was recommenced, with the same result as before. Sulphuric acid was again given, conjoined with etherial and alcoholic stimulants, which had now become indispensable, on account of the excessive faintness and weakness of pulse which had resulted from the loss of blood. On the 9th, tannic acid was prescribed, but it proved as ineffectual as the previous medicines.

The condition of the patient was now hazardous in the extreme. She was evidently sinking. The blood passed during micturition was apparently nearly as copious as the urine, and enough escaped into the bed to make it necessary to change the sheets several times a day. There was a loud diastolic murmur heard all over the central portion of the heart; and the pulse was 94, jerking and extremely weak. Another vaginal examination was instituted, but no additional light was thrown upon the source of the hemorrhage. With a vague idea that the cardiac derangement might, in some way, lie at the root of the evil, a trial of digitalis occurred to me. In such a case any chance was worth seizing; and Dr. Lee consented to the experiment. On the 10th the medicine was commenced; half an ounce of the infusion being given three times a day. On the morrow the improvement was remarkable. The discharge was comparatively scanty; the appetite had returned; and the patient expressed herself immensely relieved. During the night the discharge totally and finally ceased.

From this time the improvement was most rapid. On the 13th the digitalis was exchanged for iron; and on the 31st the patient left the hospital, tolerably strong, and declaring herself perfectly well. And there is reason to believe that she so remains; for she has never come back to the hospital, though she promised to do so at once in case of a relapse.

It now became a question whether this sudden discontinuance of the hemorrhage was to be regarded as a mere accidental coincidence, or was to be attributed to the action

of the digitalis. And supposing that the change was in consequence of the medicine, it remained to be discovered in what way the medicine operated; whether directly or indirectly; whether upon the general circulation first and secondarily upon the uterine; or whether upon the uterus especially, in some manner hitherto unexplained. It is the object of this paper to answer these inquiries.

Since the occurrence of the aforesaid case patients have been admitted into the Burton Ward labouring under menorrhagia, arising from different causes; in age they have varied from 19 to 64; and in other particulars they have differed as widely. Many of the cases have been of a kind to get well without much medical interference; these have always been allowed to do so. In every case requiring active remedies, admitted since October, 1854, the administration of digitalis has constituted the sole treatment. The discharge has invariably been extinguished; the time of its cessation varying with the strength of the medicine. In cases where the medicine was given in the largest doses, the discharge has not appeared after the second day; where the remedy has been given in a more dilute form, the hemorrhage has never outlasted the fourth.

The effects which follow the administration of a large dose—an ounce and a half of the infusion, for instance, are such as to establish beyond question the powerful influence of digitalis upon the uterus. A few minutes after the draught is swallowed, the patient complains of acute pains in the back and hypogastrium, which she compares to the pains of the first stage of labour; a quantity of blood, solid and fluid, is then ejected; and the discharge is absent until, after several hours, the pain subsides, and the bleeding returns with diminished force. After each repetition of the remedy, the cessation becomes longer, and the recommencement more feeble, until after three or four doses the discharge ceases.

It is not my purpose at present to dwell upon the *modus operandi* of the drug; I merely wish to show that its effects are immediate and well marked. When a morbid action

gradually subsides, after a course of treatment, the amelioration may be quite independent of the medicine swallowed ; but when each draught is followed by a temporary removal of the disease, we have no choice but to admit that the recovery is in consequence of the treatment. A case may be here introduced which will serve to illustrate some of the preceding statements.

A married woman, named Mary Lee, 37 years of age, the mother of a large family, was admitted on the 18th of January. She gave the following account of the progress of her disorder :

“ Ever since the last labour, which occurred nine years ago, the menses have been profuse, and mostly mixed with coagula. Eleven months ago, after much hard work, with insufficient food, the hemorrhagic discharge became nearly continual : and she came to St. George’s, at first as out-, latterly as in-patient, undergoing treatment for four months without much temporary or any permanent advantage. Short irregular intervals have occurred until within the last eight weeks, during which period the discharge has been excessively copious, and has not intermitted for a single day.”

When she was admitted she was in a state of the most extreme anæmia ; the heart’s beats were very feeble, but without murmur ; the pulse was 70, and extremely weak and small. The discharge was constantly flowing into the bed ; besides which, whenever urine was evacuated, great quantities of blood gushed from the vagina. The quantity of sanguineous fluid thus voided was estimated at four pints in the day and night ; of this a great portion was urine ; but it seemed that about a quarter of the amount was pure blood.

Half an ounce of the infusion of digitalis was now ordered three times a day, in an ounce of water, commencing on the 18th. On the morning of the 22d, the discharge ceased somewhat suddenly. No other effect was observed from the medicine ; the pulse was reduced only two beats ; it remained quite regular, and of about the same character

as before. On the 24th, the medicine caused vomiting, and the pulse became reduced from 68 to 60, still remaining regular. Whereupon the digitalis was exchanged for a solution of citrate of iron.

The patient progressed favorably until the 12th of February, when a little sanguineous discharge reappeared, which was merely supposed to indicate the return of the monthly period. But on the following day the patient was fainting with loss of blood, which was pouring out with nearly its former profusion; the pulse then numbering 72. The infusion was now given at the rate of an ounce and a half three times a day. The effect of the first dose was to cause pain, which supervened in about five minutes, chiefly referred to the back, but also affecting the lower part of the belly, and said accurately to resemble pains in the first stage of labour. This lasted acutely for about half an hour, and then gradually diminished, not entirely disappearing for some hours. The accession of the pain was immediately followed by the forcible expulsion of a quantity of blood, mostly coagulated; after which the bleeding ceased, and remained absent so long as the pain endured.

Except that each recurrence of the discharge was scantier than the preceding, the same phenomena occurred after each repetition of the medicine, until the fifth dose, after which no blood was passed. The medicine was continued until the 18th, when the patient was attacked with vomiting, looseness of the bowels, and a sensation of heat about the mouth and throat; and the pulse, which had been gradually increasing in rate, was found to have reached 92. The unpleasant symptoms presently subsided; iron was prescribed; and the patient speedily acquired a condition of health to which she had been long a stranger.

A vaginal examination was now made, and the uterus was found in a perfectly natural condition. There was a little thickening of the os, such as is generally observed in women who have borne many children. It is to be regretted that circumstances prevented an examination immediately before the administration of the foxglove; but perhaps it

would not be too much to assume that the uterus was then in the same state as it was some months previously, when the symptoms were much the same. The body and neck were then so distended, and the os so open, that a fibrous tumour was suspected to exist.

After a due interval the natural catamenial discharge appeared, no coagula were passed, and the evacuation was scanty, and lasted but two days. The patient then left the hospital in a state of perfect health, and resumed her former laborious occupation. She was seen very recently (September 17th); she was perfectly well, and the menses had never shown the slightest tendency to transgress their natural limits.

It is not necessary to dwell longer upon individual cases; it is enough to repeat that, so far as regards a speedy cessation of the discharge, the result has been the same in *every* case subjected to the treatment. I should here mention that the digitalis has always been given in company with wine and good diet. Owing to the kindness of Dr. Barclay, I am enabled to confirm my assertions by the results of his practice. Dr. Barclay writes, "The cases in which I have employed it" (*i. e.* the digitalis) "were such as seemed to me to be instances simply of functional derangement, which did not require the interference of the accoucheur; and I can say with confidence, that this mode of treatment has been more satisfactory in its results than any other I have hitherto adopted. Cases, indeed, have not been wanting which have resisted the influence of tonic and astringent remedies, which have yielded readily to this."

Before quitting the first part of my subject, I may allude to the results of a few experiments upon the sanguineous discharge resulting from organic disease of the uterus. It seems that in these cases the remedy acts with somewhat less certainty than in the preceding, a larger dose is required, and the effect is sometimes transient.

In a case of polypus, where violent hemorrhages were apt to occur, one of the attacks was suddenly cut short, while at its height, by the administration of an ounce of the infusion. The discharge ceased within an hour of the taking of the first dose, and never occurred again, although the tumour was allowed to remain untouched for three weeks. Where hemorrhage resulted from a fibrous tumour distending the uterus, the same dose produced the same effect, except that it was less permanent. In a case of advanced cancer of the os uteri, with continual and profuse sanguineous discharge, an ounce and a half of the infusion was found to cause total cessation of the hemorrhage within a quarter of an hour, the suspension generally lasting for about twelve hours. This sequence was observed as often as the experiment was made. In the same case it was found that half an ounce of the same preparation was perfectly ineffectual. Where severe attacks of bleeding depended upon rigidity of the uterus, with a roughened and tubercular state of the os, the hemorrhages were found to cease after five or six half-ounce doses. The advantage here was merely temporary.

Taking into consideration the preceding facts, I think we are justified in believing that digitalis cures, with more certainty than any remedy hitherto employed, cases of menorrhagia not connected with organic disease; and that, where organic disease gives rise to the symptoms, the action of the medicine is scarcely less manifest, although the advantage may be temporary.

I shall now consider the *manner* in which digitalis influences the uterus so as to produce these effects. The alteration must depend on some change, either in the local organism or in the general circulation. That the change is not in the latter is evident from the fact, that hemorrhages from other parts of the body are not controlled like those from the uterus. Moreover, it was found, that on the cessation of the hemorrhage, the pulse was not uniformly slower or weaker than when the treatment was com-

menced. It was found to have become slower in only seven of the sixteen observations, and even then the retardation was generally very slight. Then as to the *force* of the circulation, the pulse was not found to have become perceptibly weaker, except in one solitary instance. Thus it seems that the peculiar uterine influence is quite independent of any change in the heart's action or general circulation. It must, therefore, depend on some change wrought by the medicine in the local organism.

As far as I know, there are but two kinds of action which could be thus established in the uterus which would serve to account for the phenomena. Contraction of the capillary vessels, or the firm closing up of the uterus itself, might either of them prevent the effusion of blood from the lining membrane.

That constriction of the vessels is the change induced is a theory which cannot be upheld. Such an effect would probably not be confined to the uterus, but would be equally observed in other parts of the body. Hemorrhage from the lungs, bowels, and kidneys, would be amenable to the remedy. Besides this, it will be seen that there are certain particulars in the effect of the medicine which are not compatible with the supposition.

All the facts point to the conclusion that the *digitalis* acts by causing muscular action in the uterus itself. A short time, generally about ten minutes, after a considerable dose of the medicine is swallowed, the patient complains of severe pain in the region of the sacrum, which passes into the hypogastrium, and in every respect resembles the pain of the first stage of labour; very shortly after this a considerable quantity of blood, generally partly coagulated, is forced out of the uterus. No more discharge is then observed for several hours, so long as the contractile pain continues; and the same effect is noticed after each subsequent dose until the hemorrhage is abolished. In the case of Mary Lee, given at page 5, this succession of events was observed four times. Other cases have occurred at the hospital, in which the same effects were noticed.

A patient, named Ellen Morling, who was affected with very severe uterine hemorrhage, was ordered an ounce and a half of the infusion three times a day. The first dose was taken in the evening; very shortly afterwards pain came on in the back, and along the inside of the left thigh, precisely resembling, in character and position, the pain of her last labour. Within fifteen minutes of the taking of the medicine, a clot, said to have been as large as an egg, was expelled, and the discharge totally ceased until the following morning, when it recommenced. Another dose was then administered, and the same results were observed to follow with more quickness; a mixture of solid and fluid blood had been expelled, and the discharge had totally ceased, within five minutes. The discharge reappeared a short time before the third dose was given in the afternoon. The effects were the same, but more immediate; scarcely any interval was said to have existed between the taking of the draught and the expulsion of the contents of the uterus and suspension of the discharge; the pain was more severe than it had previously been. On the following morning the discharge reappeared in very small quantity; the remedy was given in half-ounce doses, and the cure presently completed.

The chain of symptoms here observed—the labour-pains, the expulsion of the contents of the uterus, the suspension of the discharge, seem to be the precise effects which might be expected to result from forcible contraction of the uterus; and it is not easy to suggest any other explanation.

A striking confirmation of this theory was supplied by a case which, at first sight, seemed contradictory. A patient, named Ann Cage, was brought into the Burton Ward, suffering from the symptoms of malignant disease of the uterus. She had a profuse sanguineous discharge. Hemorrhage, under similar circumstances, had previously been suspended by digitalis, and now the medicine was given in the same doses as before. An ounce and a half of the infusion was ordered twice a day. The first dose was ineffectual; it was repeated after two hours with no better

result. After two more repetitions of the medicine, the discharge was still flowing with unabated profusion, and it became manifest that the symptom was not amenable to the remedy. This was the first and only exceptional case. On making a vaginal examination, an irregular fungoid tumour was found attached to the anterior lip of the os uteri. This was extremely vascular, and its surface was the source of the discharge. The entire disease was external to the cavity of the uterus; the organ itself was healthy. If the medicine possessed a merely astringent action, its effect would be the same whether the cancer were within or without the cavity of the uterus.

Before concluding this portion of my subject, I may give the results of some experiments upon the recently impregnated uterus, which, if not sufficient in themselves to establish the stimulant effect of digitalis upon the organ, are valuable as tending strongly to confirm the conclusions previously formed.

E. Waters, being, as she supposed, very near the termination of her fifth pregnancy, had an attack of vague abdominal pains, which presently passed off. On the following day no pain whatever was experienced, and there was no sign of impending labour; whereupon an experimental dose of 20 drops of tincture of digitalis was administered. In fifteen minutes slight uterine pain was experienced; and in fifteen minutes more the patient was unmistakably in labour. Within nine hours the child was born. Ten minutes after that event 50 drops more of the tincture were given. This was followed, in ten minutes, by a most marked increase in the uterine pains; and for the next twelve hours the after-pains were excessively frequent and severe, more so than she had ever before known them.

Mary Macaulay was delivered of her first child on the 1st of August. After-pains were complained of on the following day. On the 3d and 4th they were completely absent. On the evening of the 4th, half an ounce of the infusion of

digitalis was given, and severe pains were felt through the night. The medicine was continued three times a day until the 7th. During the whole of that period the pains were frequent, and latterly they were very acute. When the medicine was discontinued they at once subsided.

Harriet Killick was delivered of her sixth child on the 18th of August. In previous labours she never had after-pains severely, and they never lasted beyond the second day. An hour after the completion of the labour, the patient being free from pain at the time, one ounce of the infusion was administered, and ordered to be repeated twice a day. A quarter of an hour after the first dose acute labour-pains came on, as severe as while the child was in the uterus. This subsided after two hours, and the patient was free from pain until the second dose was taken. This was followed by another attack of pain, which, however, was not so severe as the previous. When the medicine was taken for the third time, the patient had been for hours without uneasiness of any sort. In a quarter of an hour she was seized with after-pains of the same character as previously, but of much greater severity. "She thought she should have been torn to pieces." The patient now declined to take any more of the medicine, and had no further attacks of pain. It was observed in this case that there was almost a total absence of sanguineous discharge, although in all previous labours it had been profuse.

In four other cases the pain of uterine contraction was observed to depend on the administration of digitalis. The particulars are not given, as the cases very closely resemble those already narrated.

In these experiments the fact sought to be established is simply the connection between the pain of uterine contraction and the absorption of digitalis into the system. It is not expedient to multiply cases, as the experiment seems not devoid of cruelty, and, if it were recklessly enforced,

might well be productive of danger. These cases seem quite sufficient to prove that the drug has the action upon the uterus which has been previously attributed to it. It stimulates the nervous system of the organ, and excites the muscle to contract. It stimulates the ganglia in which the motor power of the uterus resides. This fact is not without significance. It may help us, eventually, to a more general and philosophical view of the action of foxglove than we have yet attained. It will be seen that this effect upon the uterus is precisely analogous to the influence exerted by the same medicine upon the heart. In a lecture given by Dr. Bence Jones before the College of Physicians, and printed in the 'Medical Times,' the action of digitalis upon that organ is very fully explained. It is therefore not necessary, in this place, to do more than repeat the conclusions there arrived at. It is there shown that the medicine acts as a stimulant upon the musculo-motor system of the heart, although that effect is at first masked by an action of the same kind upon the pneumogastric nerves. "It may safely be assumed that in man digitalis acts on the nerves that regulate the heart's action, first as a stimulant, and in large doses as a sedative."

The operation of foxglove upon the other involuntary muscular organs yet requires to be accurately observed. It is by no means impossible that its effects upon the heart and uterus may turn out to be merely portions of an action which occupies a wider field than has hitherto been supposed.

In concluding, I wish to express my regret that I am unable to give any comparative statement of the manner in which the uterus is affected by the various preparations of digitalis; but I cannot now supply deficiencies, as my opportunities for observation have drawn to an end.

I cannot sufficiently express my obligation to Dr. Lee, for allowing me to make free use of the cases in his ward.

It is to his kindness that this paper owes its existence. I am, moreover, indebted to Dr. Barclay for valuable suggestions, and to Dr. Bence Jones for invaluable counsel and encouragement.

HISTORY OF A CASE
IN WHICH
A CEDAR PENCIL
WAS LODGED IN THE CAVITY OF THE ABDOMEN
FOR EIGHT MONTHS.

BY
JOHN ERICHSEN,
PROFESSOR OF SURGERY AT UNIVERSITY COLLEGE, AND SURGEON TO
UNIVERSITY COLLEGE HOSPITAL.

Received Nov. 18th, 1855—Read Dec. 11th, 1855.

ON the 16th of May last I was requested by Mr. Bryant to see with him a young woman, 28 years of age, a governess, who had come up to London in order to have a cedar pencil, which was said to be lodged in the abdomen, extracted from that cavity. On inquiry into the history of the case, the following facts were elicited, not, however, without difficulty. The patient stated, that about the end of September, 1854, eight months before we saw her, she felt some difficulty in passing water, that, in order to relieve this, she was advised by a friend to pass a pencil into the urethra. That she attempted to do so, but, that whilst so engaged, some one happened to enter the room,—that the interruption caused the pencil to slip out of her hand, and that on sitting down shortly afterwards, she was seized with acute stabbing pain

in the lower part of the abdomen; that the pencil disappeared, and that neither she nor the medical man by whom she was shortly examined, had been able to ascertain where it was lodged. After this, she had repeated attacks of peritonitis, but that the pencil had not been found, nor had it ever come away by any of the natural channels. She has not suffered from dysuria, or irritation of the bladder, nor has blood ever been discharged from that organ or from the vagina; neither has there been any pain or uneasiness in defecation; blood has, however, been occasionally discharged per anum; but to this she paid no special attention, as she had for many years been the subject of piles, and occasionally had passed blood before the accident.

She now complains of constant pain in the abdomen, of a shooting or stabbing character; suffers from constant vomiting or retching, is debilitated, emaciated, and worn by her sufferings and the repeated attacks of abdominal inflammation, and is anxious to submit to any procedure that would free her from her misery.

Examination of the abdomen.—Mr. Bryant had, previously to my seeing the patient, detected what felt like the point of a pencil projecting directly forwards against the abdominal wall of the right side, about midway between the umbilicus and Poupart's ligament. On passing my hand over the abdomen, I distinctly felt this projection, apparently very superficial, as if it were just under the integument. It gave the sensation of the end of a cut pencil. It was distinctly moveable in a direction upwards, and could be pushed back somewhat, so as partly to disappear, but, on the pressure being removed, immediately returned to its former situation. The abdomen was swollen somewhat, slightly tympanitic, and uniformly tender; but more particularly so about and below the umbilicus, to which spot she referred most of her sufferings.

On passing a sound into the bladder, no trace of the foreign body could be found in the interior of that viscus, though, on turning the point of the instrument to the right

side, a hard body could be indistinctly felt through and outside its walls.

On exploring the vagina there was no sign of the pencil in that cavity, but, on passing the finger high up, and to the right of the cervix uteri, the pencil could be felt outside and through the walls of the vagina, lying apparently directly across the body from front to back.

On examining the rectum the same conditions were found as in the vagina, viz., the foreign body could be felt high up through its walls, lying to the right of the gut. The posterior extremity seemed to be lodged in the hollow of the sacrum, but it could not be reached by the finger.

From this examination it seemed evident that the pencil lay to the outer and right side of the bladder, vagina, and rectum, stretching across the body from the point, midway between the umbilicus and Poupart's ligament in front, to the hollow of the sacrum behind, apparently directly across the cavity of the abdomen. On further examination it was found that the position of the point of the pencil projecting anteriorly could be influenced by pressing on its posterior part through the rectum, in such a way that it would seem as if it were rotating upon a central axis. Thus if the posterior part was raised, the point became depressed; if, on the contrary, the posterior part was drawn down, the anterior extremity rose upwards towards the umbilicus; if it was pushed to the right, the other moved to the left, &c. In whatever direction, however, the anterior extremity was moved, it always came back to one fixed point,—that already indicated, about two inches above Poupart's ligament.

Mr. Bryant and I, consequently, came to the conclusion, that the pencil was lying across the cavity of the abdomen, that it was fixed about its centre by some tissue traversed by it, and that the point, which felt very superficial, was engaged in the anterior abdominal wall. What was to be done under these circumstances? Was the pencil

to be left, or was an attempt to be made to extract it? These questions were anxiously discussed by us. If it were left, there seemed little doubt that the patient would speedily die from peritonitis, of which she had had several attacks, and from which she was apparently suffering in a subdued degree, or else that she would be worn out by the continued disturbance of digestion, and consequent impairment of nutrition, kept up by the foreign body. If, on the other hand, an attempt were made to remove it, there was every reason to apprehend that inflammation of the peritoneum might be lighted up, which would probably prove fatal in the patient's weakened state. We were, however, in hopes that the track along which the pencil lay had been shut off from the rest of the peritoneum by the deposit of lymph, and that thus the danger of peritonitis would be materially lessened. However, it appeared to us that the probable danger of extraction was less than the certain danger of allowing the pencil to remain in the abdominal cavity, and we accordingly came to the opinion that its removal ought to be attempted.

Operation.—At 4 p. m., the patient having been anaesthetized, the water drawn off, and the rectum ascertained to be empty, I proceeded to operate, being ably assisted by Mr. Bryant, who, passing his fingers deeply into the rectum, pushed the posterior end of the pencil upwards and forwards, so as to make the point project as much as possible. I cut down on this through the several layers of the anterior abdominal wall, until the fascia transversalis was reached, in which the black-lead point of the pencil was fixed, and through which it projected. I now slightly enlarged the opening in the fascia, and stripping back the tissues somewhat, exposed enough of the pencil to enable me to withdraw it with a pair of necrosis forceps. On removal it was found to have a strong feculent odour, and to be deeply stained in places with the intestinal contents. It was five and a half inches in length, cut to a sharp point, which was

still perfect. The two halves of which it was composed had separated. On examining the pencil more closely, it was seen to be distinctly marked by three broad bands, which ran completely round it. Two of these, towards either extremity, were rough and dark with feculent staining; the third, in the centre, was smooth, clean, and quite free from any feculent imbibitions. From this examination it appeared as if the pencil had lodged in the interior of the intestine, and in contact with feculent matters in two situations. No feces or flatus, however, followed the extraction of the pencil, or escaped by the wound, which was closed by two sutures and some plaster.

The patient was put in bed with the knees raised, confined to ice- and barley-water, and opium was pretty freely given.

On the day following the operation, a severe attack of peritonitis came on, specially marked by incessant vomiting and retching, and by obstinate constipation. In spite of active treatment of the usual kind these symptoms continued, and the patient died on the fourth day after the operation.

Examination about fifty hours after death.—The body presented signs of commencing decomposition; the abdomen was discoloured and tympanitic. On opening the peritoneal cavity, some flatus escaped. There were several ounces of turbid, rather dark-coloured serum in the cavity of the peritoneum, with flakes of recent lymph gluing the intestines together at several points. The omentum was much injected. About midway between the umbilicus and pubes, to the right of the mesial line, a mass of small intestine (ilium) was found glued together by old and tough lymph, forming a tumour composed of several coils of intestine, and altogether about as large as the fist. This mass was generally dark coloured, of a reddish purple tint, deeply injected, and presenting on its surface deposits of recent lymph in addition to the old lymph, by which its component coats were

matted together. On attempting to separate the coats of which this mass was made up, some fluid feces and air bubbled out from between them, and on examining the source from which this came, it was found that two of the coils of intestine had been perforated, each in two places. In one, the apertures were close to each other, at the mesenteric border of the intestine. In the other, they were nearly opposite to one another, traversing the centre of the gut. These apertures were all rounded, smooth, and exactly of the same size and shape, corresponding in these respects to the pencil, by which they had been made. They were surrounded by a quantity of old and dense lymph, by which the neighbouring portions of intestine were so firmly attached to one another, and to the wounded gut, as to be separated with difficulty. The bladder, uterus, and rectum were sound.

On examining the vagina, a depressed cicatrix was found at its upper and posterior cul de sac, close by the side of the uterus. The peritoneal aspect of this cicatrix was thickened, puckered, and depressed.

From this it would appear that the pencil perforated the posterior wall of the vagina, passing upwards behind the bladder, and then traversing the peritoneal cavity. There can be little doubt that this accident happened whilst the patient, disturbed during her attempts to pass the foreign body into the urethra, allowed it to slip out of her grasp, and then suddenly sitting down, forced the pointed end of the pencil into the abdomen. She was seen and carefully examined a few hours afterwards by a medical practitioner of the town in which she was at the time residing, and he was unable to find any trace of the pencil, which he certainly would have done had it been lodging in the cavities of any of the pelvic organs. At this time, also, the intestine was doubtless perforated, and continued transfixed through two of its coils until the time of extraction, a period of nearly eight months.

Not the least interesting feature of this case is the absence of all feculent extravasation into the cavity of the peritoneum, not only during the sojourn of the pencil within the abdomen, but after its removal. Whilst in the abdomen, and firmly fixed in the intestine which it traversed, the non-escape of feculent matters might be accounted for by the pencil, mechanically as it were, blocking up the apertures it had made; but after its withdrawal, the absence of extravasation must, I think, be accounted for by the influence of the uniform pressure of the abdominal organs on one another.

WOUND
OF THE
ABDOMINAL PARIETES;
PROTRUSION OF OMENTUM AND TRANSVERSE COLON;
DIVISION OF THE LATTER TO THE EXTENT OF
FOUR FIFTHS OF ITS CIRCUMFERENCE:
RECOVERY.

BY
NATHANIEL WARD,
ASSISTANT-SURGEON TO THE LONDON HOSPITAL.

Received Nov. 21st.—Read Dec. 11th, 1855.

ON the morning of Sunday, September 30th, in the absence of Mr. Luke, I was called by Mr. Austin to visit a healthy woman, æt. 51, an inmate of Bethnal Green Asylum, who had, during the night, inflicted on herself a severe wound in the abdominal parietes with a common razor. I found an irregularly zigzag incision, five inches in length, with the centre of it crossing the direction of the right epigastric artery. It was distant four inches from the umbilicus, and was directed obliquely from below upwards and outwards in the hypogastric and umbilical regions. From this wound protruded omentum and a portion of the transverse colon. The former was bruised, and detached superiorly somewhat from the gut; the latter had been cut

through, with the exception of one fifth of its circumference, the undivided portion remaining in connection with the meso-colon. The mucous membrane of either nearly separated portions pouted out so as to conceal from view the muscular and serous tunics. Blood was effused into the peritoneal cavity, but fecal extravasation, either inside or outside of the cavity, was not observed. To the inside of the protrusion the skin and fascia had been detached as far as the linea alba, so that the hand could be placed between them and the abdominal muscles. The muscles around and behind the protrusion were irregularly cut and jagged, and the opening into the abdominal cavity was about the size of a five shilling piece, more vertical in direction than the superficial wound, and about an inch nearer to the umbilicus. The woman was blanched, her feet were cold, and her face was bedewed with a clammy perspiration. She complained of great thirst. I stitched up the wound in the colon with the uninterrupted suture, which was passed through all the coats of the intestine, first from within outwards through one lip of the nearly divided gut, and then from without inwards through the other lip; and so on, alternately, until the two lips were brought into close approximation with each other. A small round sewing needle was used, the ligature being knotted at the end. This knotted end, from the manner in which the suture was applied, remained in contact with the free surface of the mucous membrane. The lips of the wound having been brought into close apposition, the thread was finally knotted on the outside of the serous covering of the gut, and the thread cut close off from the knot. Thus one extremity of the silk ligature was within, the other outside the bowel. The intervals between the stitches were about the sixth of an inch from each other, and about the same distance *through all the tunics*, from the border of the lips of the wound. Here and there, where the outline of either lip had been somewhat jagged and irregular, the mucous membrane bulged up from below. The mucous, muscular, and serous coats of the two segments of the bowel were thus brought into as

close mutual contact as could well be effected, considering the circumstances of the case. With a little careful manipulation, I reduced the protrusion within the abdominal cavity, and then applied the uninterrupted suture to the wound of the peritoneum, leaving, however, unclosed a small opening below, equal in size to the tip of the middle finger. The epigastric artery, which was not bleeding at the time the bowel was being stitched up, now required a ligature. One suture only was applied to the wound in the integument, one or two small partially detached portions of the external oblique tendon having been previously removed with the scissors. The abdominal walls were supported with strips of lint, strapping, and bandage.

The operation, during which brandy and ammonia were given at intervals, was necessarily very tedious, two hours having gone by before it was entirely completed. The patient scarcely moved or uttered a word the whole time. After she had somewhat rallied, she gave the following account of how and why she inflicted the injury.

“When on a visit to her home, on the 20th of September, nine days before her attempt at suicide, she took a razor from her effects, and concealed it in her bustle. At 11 o'clock, in the night of the 29th, she got out of bed, made water, took the razor, which had been sewn up in her bustle, got into bed again, said prayers, and having asked God to receive her soul, began to cut herself, which hurt her very much. She continued to mangle herself all night more or less. Finding that she did not die, she turned over on her stomach, in the hope that the blood gravitating would the sooner put her out of her misery. She made the final gash when it was light in the morning.” At the hour of rising she excused herself for not getting up by stating that she was ill. Shortly after this she got out of bed, looked in the glass, and smoothed her hair down. On the attendant coming, at 20 minutes past 8 o'clock, with her breakfast, she said she had killed herself; and then the injury and razor were discovered.

On Mr. Austin seeing her, she expressed her regret that

she had not killed herself, and said, that certain of going to heaven herself, she should meet no mad doctors there, as hell was their doom. Beyond this style of talk she was perfectly rational and coherent. She expressed an earnest wish to see her son, and particularly desired that her attempted suicide might be concealed from him.

The plan of constitutional treatment that I recommended to be adopted, was approved of by Mr. Austin. It was to place the patient under the full influence of opium, and to allow little or nothing to be taken into the alimentary canal for some time. Ice was prescribed, and, at 2 p.m., P. Opii, gr. iij, were given in powder, and were immediately rejected; 3 p.m., another dose of the same quantity was retained; 6 p.m., another three-grain dose was given, and repeated at 12 p.m.

Mr. Luke, who subsequently attended the patient, fully agreed in the propriety of carrying out this treatment.

Oct. 1st, 7 a.m.—Patient has passed a quiet night, free from pain. The nurse's back being turned, she got out of bed to make water. Surface warm; pulse 90, not very feeble; tongue clean; countenance not anxious; discharge rather fetid. To have lumps of ice, and nothing else. Six grains of opium were given to-day in two doses.

2d.—Has slept a good deal since yesterday; passed a good night, with occasional starting from sleep, the result, as she says, of abdominal pain. She is restless, and attempts to justify herself; is telling lies of everyone around her, and falsely accusing the attendant of cruelty, &c. P. Opii, gr. iij, 11 a.m.; P. Opii, gr. iij, 5 p.m.; and P. Opii, gr. iij, 10 p.m.

3d.—Quite free from pain; pulse fuller, soft, 90. Complaints of hunger: to have nothing but ice. P. Opii, gr. iij, 10 a.m.

4th.—Hardly so well; expression anxious; no pain; pulse 112, feebler. P. Opii, gr. iij, at noon; 8 p.m., P. Opii, gr. iij; midnight, Liq. Opii, ℥xl. To have strong beef tea, cool.

5th.—Expression less anxious, more comfortable men-

tally; no pain or abdominal distension. Beef tea, with bread crumbs. Liq. opii, $\mathfrak{m}\mathfrak{j}$, at bed-time.

6th.—Has passed a rather restless night. Pulse 94; tongue not dry, slightly coated, and brown; a slight erysipelatous blush extending towards the right axilla. Takes strong beef tea, toast, and iced-water. Soap suppository; Liq. Opii, 3j, at bed-time. There is a small abscess in the abdominal wall, at the upper extremity of the wound.

7th.—Passes flatus per anum; no abdominal pain or distension. An injection of half a pint of gruel was immediately ejected, in consequence of the perversity of the patient.

8th.—Pulse 96, fuller; slight flatulent distension; tongue furred and brown; wound rapidly closing; discharge rather fetid. An ounce of castor oil, taken at 9 in the morning, not having acted, the same dose in half an ounce of brandy was repeated at 2 in the afternoon. At 7 p.m. the bowels acted, and the motion was copious and solid.

9th.—Bowels open three or four times up to 4 p.m., the last motion quite liquid; pulse 88; tongue coated; wound healthy. Haust. Rhei. c. Tinc. Opii, 3ss, checked the purging.

10th.—The tongue cleaner; bowels open; skin cool; a narrow slough from the upper extremity of the wound was removed. Liq. Opii, $\mathfrak{m}\mathfrak{x}\mathfrak{l}$, at bed-time. On the 13th, the ligature from the epigastric artery, and on the 14th, that from the peritoneum, came away.

23d.—The wound is reduced to a narrow line of healthy granulations.

30th.—Wound all but healed; no medicine has been required, but an opiate at night. During the last fortnight has had a mutton chop, fish, and three ounces of brandy daily. The bowels have been regular.

The wound had healed on the following day; and on the 2d of November the patient got up, and has been up and active ever since. Thus the abdominal wound had healed on the thirty-second day after it had been inflicted. The ligature was not discovered.

Remarks.—For the greater part of the details of this case, I am indebted to my friend Mr. Austin, who watched the patient with unremitting attention, and carefully registered, day by day, anything worthy of mention during her progress towards recovery. The case affords, I think, an admirable illustration of the main principles of treatment, local and constitutional, that should be had recourse to in instances of wound of intestine that are similar to it in character and extent. I have submitted it, however, to the consideration of this Society, principally with the object of adding to the somewhat scanty evidence already on record, that tends to substantiate the conclusions first inductively arrived at by Mr. Travers,¹ in reference to the proper surgery of these severe but interesting injuries. Excluding from consideration the elaborate nature of the wound in the abdominal parietes, it would be difficult to conceive a better case for testing the validity of the plan of proceeding recommended by him. The good health of the patient bodily, her peculiar psychical condition, and the clean cut that had been made in a part of the intestine, of less vital importance in the œconomy than the small, and the absence of fecal extravasation, were conditions that predisposed to its successful termination. This favorable termination was, no doubt, further ensured by the adoption of the opium treatment, and the temporary abstinence from food, both of which had been enjoined with the view of suspending for a time the peristaltic action of the alimentary canal, until it had become probable that the wound in the colon had been bridged over by a firm uniting medium.

For the first four days the patient took nothing but ice and opium; and the quantity of the latter administered during the first five days amounted to thirty-three grains. Her bowels were thus kept confined until the ninth day, on which, in consequence of flatulent distension, it was deemed proper to administer injections and castor oil, which acted well, without pain or subsequent inconvenience.

¹ 'An Inquiry into the Process of Nature in repairing Injuries of the Intestines,' &c. London, 1812, p. 188 et seq.

The absence, also, of any symptoms that could be construed into peritonitis was, it is reasonable to infer, attributable in no small degree to the free use of this valuable drug.

The correct constitutional treatment of serious abdominal lesions cannot, I think, be well overrated, and I have appended the details of a case, kindly furnished to me by my friend Dr. Wight, the East Indian Botanist, in which case a narcotic and sedative plan was effectually had recourse to.

"Some years back I was garrison assistant-surgeon," says Dr. Wight, "at Nigapatam, on the Coromandel Coast. At Naggor, a small sea-port town, about five miles from my station, there is what in that part of the country is esteemed a very sacred Mahomedan shrine or temple. A fanatical Mussulman Sepoy, probably smarting under the disgrace of punishment, took it into his head to get to heaven by a near cut, by committing suicide in this temple. With that view he obtained leave of absence from his corps, and, during one of the festivals of several days' continuance, travelled eighty miles, so as to arrive about the great day of the feast. Having said his prayers, and strengthened his resolution by a large dose of bang, he plunged a knife into the hypogastrium, and ripped up his abdomen to near the pit of the stomach. He then plunged it into the right lumbar region, and carried it across to the other, thus forming an extensive crucial opening. He quietly laid himself down to die, and in that state was left undisturbed by the bystanders. This took place about 3 p. m. He was allowed to remain in that state, the whole bowels protruding, until about 9 in the morning, an interval of eighteen hours, when one of his friends and the police thought it necessary to bring him to hospital. He was placed on a stretcher, with nothing but a thin cloth thrown loosely over him to protect him from the sun, and carried to Nigapatam, where he arrived in about two hours, still under the influence of the bang. The stomach, nearly the whole of small intestines, and transverse colon, were then protruding, and,

judging from their bright red colour, in a state of intense inflammation. In the stomach there was a wound large enough to admit the point of my finger, but otherwise the intestines were uninjured.

“The treatment was most simple. The protruding parts were gently sponged with warm water to remove clots of blood and dust; they were then returned, and the flaps of the abdomen brought together, and kept there by the interrupted suture, and the whole covered with cloths, and kept constantly wetted for some days with cold water. The only ingesta allowed for the first three days were an occasional teaspoonful of lemonade to relieve thirst, and a few enemata of barley-water. Opium was given. Under this treatment the man recovered without a single untoward symptom, and in about a month left the hospital and rejoined his regiment.”

C A S E
OF
DETACHED PIECE
OF
ARTICULAR CARTILAGE
EXISTING AS A LOOSE SUBSTANCE IN THE
KNEE-JOINT.

BY
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Received Nov. 22d.—Read Dec. 11th, 1855.

THE following case, unfavorable in its result, I am wishful to put on record, on account of its important pathological bearing.

John W—, brewer, æt. 37, of robust frame, but a free liver, became an out-patient of the Leeds Infirmary, in June, 1855. He stated, that a year ago he accidentally let a cask, which he was lowering into a cellar, slip against the right knee. Severe pain in the joint followed, and he was unable to work for three weeks; after which time he followed his employment as usual for nearly twelve months, until a few days ago, when he became suddenly lame, and was unable to bear his weight upon the right leg. He then, for the first time, felt a flat substance moving about in the knee-joint.

For six weeks he attended the hospital as an out-patient, and was often seen by my colleagues, Mr. Smith and Mr. Samuel Hey. During this time he was frequently attacked by sudden pain and lameness, being unable to walk, until the substance was moved into some position where it ceased to annoy him. We could distinctly feel the flat substance, about the size of a shilling. It could be readily pushed upwards along the outer side of the joint, then under the extensor tendons, and down the inner side of the joint. It could, again, with equal ease, be made to take a retrograde course, and resume its old place. Very little pain was produced by these forced movements. At times, when the loose body was favorably placed, the patient could walk pretty well, but on slight changes in its position, extreme pain and lameness would result. Rest and bandaging were the chief means of treatment adopted, but without any beneficial result; for, although the substance could be readily placed in a favorable position, yet the bandaging, however carefully employed, failed to diminish materially its mobility.

At length, after urgent and repeated requests on the part of the patient, I agreed, with the consent of my colleagues, to remove the loose body; for which purpose he was admitted into the hospital.

August 1st, at noon, the patient was brought into the operation-room. The knee was but slightly enlarged, and did not show any synovial effusion. The substance being pushed to the outer side of the joint, so as to rest upon the outer condyle, a subcutaneous incision of the synovial membrane was made over it, in the hope that it might then be pushed into the areolar tissue; but this was found to be impracticable, on account of its large size and irregular border. The skin was then drawn forwards, and divided over the substance, after which it was easily removed. The wound was carefully closed by adhesive plaster and a compress of lint, and the joint covered with wet linen. The limb was kept perfectly motionless by a splint placed along the back of the thigh and leg.

The substance, on being examined, was found to be flattened, circular in form, and irregular or ragged at its border. One of its surfaces had the appearance of cartilage, and was smooth and slightly convex; the other was concave and rough, from a layer of bone.

The operation, unfortunately, was followed by inflammation of the joint and phlegmonous erysipelas of the thigh and buttock, which proved fatal on the sixth day.

The limb was examined after death, and considerable infiltration of the integuments of the thigh and buttock was found; but there was not any effusion observable in the tissues immediately surrounding the joint. On opening it, a small quantity of turbid synovia was found. At the under surface of the inner condyle the articular cartilage showed a circular depression, about the eighth of an inch in depth, having a rough surface of bone at its base. On comparing this breach in the articular cartilage with the substance which had been removed, they were found to correspond accurately with each other; and, on placing the detached substance in the cavity in the condyle, the continuity of the articular surface was perfectly restored. It is therefore evident that the loose body was a portion of the articular cartilage along with a thin layer of bony substance.

The explanation, which I venture to offer of this remarkable fact, is that a portion of the articular cartilage, and of the adjoining layer of bone, had been injured by the accident which the patient sustained when moving the cask; and that, by a slow process of exfoliation, extending through a period of about twelve months, the injured part was cast off and became loose in the joint, causing the pain and lameness which led him to seek for relief.

I am indebted to Mr. Sly, one of the resident assistants of the hospital for the notes of this case, and for his unremitting attention to it during its course.

HOURLY
PULSATION AND RESPIRATION
IN HEALTH.

WITH
TWO DIAGRAMS AND TABLES.

BY
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OF THE CHEST, BROMPTON, ETC.

Received Nov. 27th, 1855.—Read Jan. 8th, 1856.

THE following investigation was made in order that I might obtain a basis of comparison for a similar inquiry in reference to consumption. Hitherto no observer has recorded the rate of the pulse and respiration in each of the 24 hours, and the relations of those functions to sleep, food, fasting, sunlight, and other economic questions. The diurnal revolutions of the pulse have been noted hour by hour by Drs. Robinson, Falconer, Knox, and Guy, for the greater part, but not the whole, of the day; but no one has performed the like duty in respect of the respiration. The two latter observers, especially, knew well the influence of posture, food, rest, period of the day, and sleep on these

functions; but it was yet a desideratum to make such inquiries on other persons than the observer, on a larger scale, and also under the conditions of food, rest, and sleep, which are maintained by a majority of the people of civilised nations, during each hour of succeeding days and nights.

The following observations were made under the last-mentioned circumstances, with the utmost regularity, and were noted in the lying, in preference to the sitting posture, as being the only one possible during sleep, and that the least likely to be influenced by the wear and tear of the day. The hours of rising and retiring to rest, and of taking meals, were those adopted at the Hospital for Consumption: viz., immediately after 8 a.m. and before 9 p.m. for the former, and 8½ a.m., 12½ to 1 p.m., 5½ p.m., and 8½ p.m. for the latter.

The subjects were the members of my own family: viz., two ladies, æt. 33½ and 39, my two female children, æt. 6 and 8½ respectively, and myself, æt. 36. All were of active habits, and excitable temperament, and free from any hereditary or other disease.

In the examination each one lay down five minutes before the hour, and was examined in regular order. The pulse was counted in half minutes, and the respiration in whole minutes; and the counting was commenced only from a long line on the dial.

Two sources of error were observed in both pulsation and respiration. In pulsation—1st, the disposition to intermit, chiefly in the children, and, in all, when the pulse was low, as before a meal, at the close of the day (especially in the standing or sitting postures), and during the night. 2d, the influence of yawning and other irregular and intermittent respiratory efforts, each of which caused an after but temporary increase of pulsation. This induced constant irregularity of the pulse when the person was fatigued. In respiration—1st, from its shallowness directly after a meal, late in the evening, and early in the morning before awaking. 2d, from mental action and other influences during the day,

causing constant variation in the number of respirations. The pulse was most uniform during the day; the respirations most uniform in the night. All these difficulties were overcome; but they served to prove the dependence of the pulse upon respiration, and the influence of breathing in maintaining the circulation of the blood.

Diagrams Nos. 1 and 2 represent the results obtained, the former for each hour of the three days, or 72 hours, of the inquiry; and the latter the average of each hour in the day, for the same period; and also the returns from a prolonged fast.

I shall first consider the rate of pulsation and respiration absolutely, and then as influenced by disturbing causes.

A. RATE OF PULSATION AND RESPIRATION ABSOLUTELY.

The average rate may influence the hourly changes of the rate of these functions. It was as follows:

TABLE No. I.

	Æt. 6 years.	Æt. 8 years.	Æt. 33 years.	Æt. 36 years.	Æt. 39 years.
Pulsation . .	94·2	80·0	73·4	72·2	61·0
Respiration .	20·6	20·8	18·3	17·8	17·8

It thus was inversely as the age, and much more so in the youngest (æt. 6); but slight differences of age had far less influence over respiration than over pulsation.

The general average course of daily variation of the pulse was as follows: It was the lowest during the night, and the

highest during the day. It fell in the evening, before and after going to bed, and rose in the morning, before and after awaking and rising. During the night hours it varied but little; but during the day it varied greatly, for it rose directly after and fell before each meal, and, consequently, there were four elevations and depressions, as there were four meals. The greatest rise of pulsation was from before waking to the hour after breakfast; and the greatest fall was from supper to the second or later hour of sleep. The lowest pulsation was from about 1 to 3 a.m.; and that at 8 a.m. was not only the lowest of the day, but was nearly as low as that of the night. But between 5 and 8 a.m. there was commonly a considerable elevation of the pulse, enduring for two or three hours.

The respirations followed the same course, but they were even lower proportionately at night than in the day; and the evening fall was sooner attained.

Each case and age had its own peculiarity, as shown by the diagrams, but each one has about 6 periods of elevation and depression of pulsation in the 24 hours.

Thus in all the cases there was a minimum pulsation at 8 a.m., and also at midday. There was also one at 4, 5, or 6 p.m., and again at 7, 8, or 9 p.m. In many there were others at 11 or 12 p.m., or 1 a.m., and at 3 or 4 a.m. The maxima were at 8, 9, or 10 a.m., 2 or 3 p.m., 6 or 7 p.m., 11 or 12 p.m., and 4, 5, 6, or 7 a.m.; and the two former were the greatest. All these would probably change if the hours of rising and retiring to rest, and of meals, were changed.

The respiration had similar changes, but the elevation of pulsation before waking in the morning had not a corresponding rise of respiration.

*Increase of Maximum over Minimum Pulsation and
Respiration.*

The minimum pulsation and respiration varied from 52 and 14, to 85 and 16, and the maximum from 70 and 20 to 108 and 29 per minute. The excess of the maximum over the minimum varied from 14 to 34 pulsations, and 4 to $14\frac{1}{2}$ respirations; and the ratio of that excess varied from $\frac{1}{4}$ to $\frac{1}{1\cdot8}$ of the minimum pulsation, and from $\frac{1}{2\cdot9}$ to $\frac{1}{1}$ of the minimum respiration.

The average minimum pulsation and respiration was 69 and $15\frac{1}{2}$, and maximum $85\frac{1}{2}$ and $22\frac{1}{2}$; and the difference, 16 pulsations and 7 respirations, or nearly $\frac{1}{2}$ of the minimum respirations, and $\frac{1}{4}$ of the minimum pulsations.

The average proportionate increase of the maximum over the minimum, in each person, was as follows :

TABLE No. II.

CASE.	Æt. 6 years.	Æt. 8 years.	Æt. 33 years.	Æt. 36 years.	Æt. 39 years.
Pulsation . .	$\frac{1}{4\cdot6}$	$\frac{1}{2\cdot9}$	$\frac{1}{4\cdot1}$	$\frac{1}{5\cdot8}$	$\frac{1}{4\cdot5}$
Respiration .	$\frac{1}{1\cdot5}$	$\frac{1}{1\cdot6}$	$\frac{1}{2}$	$\frac{1}{3\cdot7}$	$\frac{1}{4}$

Thus the increase was exceedingly great in the respiration, and much greater than in pulsation; and the variation in the proportion of both functions was very great; but the variation of the maximum and minimum rate from day to day was but slight. The case having the largest numerical increase of pulsations had not the largest increase of respirations.

Ratio of Respiration to Pulsation.

This is highly important, and was very variable. It was the lowest in the youngest, and the highest in the oldest person, yet not in precise proportion to age. It was on the whole average: in æt. 6, as 1 to 4·5; in æt. 8, as 1 to 3·9; in æt. 33, as 1 to 4; in æt. 36, as 1 to 4·1; and in æt. 39, as 1 to 3·4.

It was identical from day to day in the oldest, and varied the most in the youngest; and it was also the least in the children.

The variation in the ratios, as noticed from hour to hour, on the average, is great and remarkable, and of great practical and physiological interest. It is greater in early life. The ratio is always greater during the day than the night, as shown in the following table:

TABLE NO. III.

Ratio of Respiration to Pulsation at each hour (lying posture).

CASE.		HOUR.																							
		Midnight.											Midday.												
		12	1	2	3	4	5	6	7	8	9	10	11	12	1	2	3	4	5	6	7	8	9	10	11
Æt. 6 years, 1 to	5·5	5·4	5·1	5·5	5·3	5·3	5·3	5·0	4·4	4·1	4·1	3·9	3·9	4·0	3·9	3·8	4·1	4·1	4·1	4·4	4·3	4·2	5·6	5·7	
" 8 "	4·1	4·4	4·3	4·2	4·4	4·1	4·5	3·7	3·3	3·9	3·2	3·3	3·3	3·6	4·0	3·4	3·6	3·6	3·7	3·9	3·5	3·8	4·3	4·0	
" 33 "	4·5	4·3	4·4	4·4	4·3	4·9	4·6	4·6	4·5	4·0	4·0	3·4	3·7	3·7	3·9	3·8	3·7	3·7	3·2	3·3	3·4	3·8	4·2	4·2	
" 36 "	3·8	4·2	4·0	4·0	3·7	4·1	4·2	3·9	4·2	3·9	4·0	3·8	3·8	4·0	4·3	4·1	4·2	4·2	3·7	4·4	3·9	3·8	4·0	3·9	
" 39 "	3·6	3·3	3·7	3·7	3·7	3·9	3·5	4·0	3·3	3·4	3·3	3·3	3·2	3·2	3·2	3·1	3·0	3·4	3·0	2·9	3·1	3·4	3·6	3·5	

Thus the highest ratios are limited to the hours of waking, and the lowest to those of sleep, and the change in the ratio is the greatest at the hours when sleep and waking join each other. This is indisputable in æt. 6 and æt. 8, and is manifest in all but myself. The extremes were 1 to 2·9 and

1 to 5·7, or the larger was the double of the lesser ratio. The hours of highest ratio were, in æt. 6, 3 p.m. (1 to 3·8); in æt. 8, 10 a.m. (1 to 3·2); in æt. 33, 6 p.m. (1 to 3·2); in æt. 36 (without sleep), 4 a.m. and 6 p.m. (1 to 3·7); and in æt. 39, 7 p.m. (1 to 2·9). The hours of lowest ratio were, in æt. 6, 11 p.m. (1 to 5·7); in æt. 8, 6 a.m. (1 to 4·5); in æt. 33, 5 a.m. (1 to 4·9); in æt. 36 (without sleep), 7 p.m. (1 to 4·4); and in æt. 39, 7 a.m. (1 to 4). Other hours had almost the same ratios. It was temporarily lessened at 4, 5, or 6 a.m., by the temporary increase of pulsation at those hours, without a corresponding increase of respiration.

It is affected by meals, but differently in different cases, being sometimes increased, but more commonly decreased, directly after a meal. If the day be equally divided at the hours after 8 a.m. and before 9 p.m., the ratios of the day and night are as follows:—æt. 6, as 48 to 62; æt. 8, as 42 to 49; æt. 33, as 43 to 52; and in æt. 39, as 38 to 43; whilst in my own case, from the absence of sleep, it was equal, or rather less, for the day, as 47 to 48. There was a gradual increase from the middle of the night to the full day; and a decrease in the afternoon, but more particularly at 10 p.m. to the depth of night.

The ratio is rather dependent upon respiration than upon pulsation; and it is not commonly greater with increased or less with diminished pulsation, or the contrary. The high ratio of the day is chiefly due to the disproportionate increase of respiration; and the low range of the night to the disproportionate decrease of that function. The relation of ratio to age is that of pulsation to age.

Thus no one ratio is indicative of health under all circumstances; but whatever may be the vital requirements for both functions, they are less fulfilled during the night than during the day.

B. DISTURBING INFLUENCES.

1. *Period of the Day.*

This is important, not in itself, but by localising influences and bounding results. Four periods occur naturally, viz.,—1st, the minimum period, or “night,” from 1 to 5 a.m.; 2d, the maximum period, or “day,” from 9 a.m. to 9 p.m.; 3d, the “morning” ascent, from 5 to 10 a.m.; and, 4th, the “evening” descent, from 9 p.m. to 1 a.m.

1st. “Night.” The total averages were, pulse 72, respiration 16·3; and the total average range of pulsation was 6·6, and of respiration 1·1. Thus the total range of pulsation was much greater than that of respiration, considered in the proportion of the absolute rate of each function.

2d. “Day.” The total average of pulsation was 77·6, and of respiration 20·8; and the range of both functions was, pulsation 13·3, and respiration 3·9. Thus all the numbers have increased from the night, and especially of respiration, so that the increase in the rate and range of that function was much greater than of pulsation, and at this period was disproportionately large. In æt. 8 the increase of the “day” over the “night” was more than 6 respirations per minute.

3d. “Morning.” The total averages were, pulse 76, and respiration 18; and the range of pulsation was 13·6, and of respiration 5·6. The average rate was thus between that of the “day” and “night,” but the range (or amount of variation) was very much greater, so much so that it was twice as great as that of the “night” in pulsation, and five times as great in respiration, and one half greater in respiration than during the “day” of 13 hours. In æt. 33 there was the anomaly of the “morning” range of respiration being less than the “night” range, and the “day” range of pulsation being less than the “morning” range. This was owing to a remarkable increase from 4 to 7 a.m. (See Diagram No. 2.)

4th. “Night.” The total average of pulsation was 74, and of respiration 17·4; and the average range of the functions

was of pulsation 7·3, and of respiration 2·6. Thus all the numbers were below those of the "day" and "morning," but the fall of the respiration from the "day" to the "night" was disproportionally great as regards pulsation. *Æt.* 36 and *æt.* 39 were exceptional, in having more frequent "evening" than "morning" respirations.

Thus, by way of summary, both functions became more frequent, on the average, through the "night," "evening," "morning," and "day," in their order. The two functions did not correspond with each other in reference to their range, but that of pulsation increased through the "night," "evening," "morning," and "day," in their order; whilst the order of increase in reference to respiration was "night," "day," "evening," and "morning." Both functions were at a minimum in the night, and a maximum in the day,—the term day and night being now taken in their ordinary sense; but it is worthy of note how great is the minimum of respiration in the night, and the maximum during the day.

2. *Influence of Food.*

This is twofold—1st, immediate; 2d, more remote. There were two sets of inquiries:—1st, for the remote, hour by hour, as already stated; 2d, for the immediate, every 5 minutes, from the commencement of mastication.

1. *The remote.*—The effect was estimated by taking the difference between the numbers ascertained before the meal, and the highest which soon followed the meal. This is liable to two fallacies:—1st. The more the system was emptied of food (within limits), the greater doubtless would be the effect of the food; and the more frequent the meals (within limits), the less would be the effect of the food. I do not know the degree in which the system wanted food at the hours indicated, and it is unlikely that the same degree would exist even in the same person at the same meal on successive days, much less at different meals and in

different persons. The frequency of meals must be noted with the result; and that frequency was much in accord with popular habits; and thus, if the full effect is not shown, the ordinary effect will be. 2d. In the interval between the examination before breakfast and that after breakfast, there was also the performance of the acts of rising and dressing; but I have proved these to be nearly null by making an examination, on October 27th, when they had taken breakfast, at the usual hour, in bed.

The meals were all simple, moderate, and without stimulants. The food for breakfast was milk, coffee, egg, bacon (or some of them), and bread; for dinner, animal, vegetable, and farinaceous food; at tea, bread and butter; and at supper, milk or oatmeal gruel; the aim being to assimilate the habits to those enforced at the Hospital for Consumption, and maintained by popular practice. The examination was not at and directly after the full hour, but about the middle of the hour after the commencement of the meal. Thus the expression "hour after the meal" does not mean a full hour, but "during the hour."

PULSATION.

TABLE No. IV.

A. *Breakfast, 8½ a.m., with the Amount and Hour of greatest increase of Pulsation.*

	Æt. 6 years.		Æt. 8 years.		Æt. 33 years.		Æt. 36 years.		Æt. 39 years.	
	Pulsa- tion.	Hour.	Pulsa- tion.	Hour.	Pulsa- tion.	Hour.	Pulsa- tion.	Hour.	Pulsa- tion.	Hour.
1st, 2d, and 3d day . . }	17, 14, 18	1, 2, 1	14, 17, 19	1, 1, 2	16, 12, 21	2, 1, 1	6, 22, 11	1, 2, 1	6, 17, 14	1, 2, 1

Thus the effect varied much, viz., from 6 to 22 pulsations, and was the least at middle life. The averages were, 14·6, 16·6, 16·3, 13, and 12·3 = 15 medium. This large total average may be due to the long absence of food. In two

thirds of the observations the maximum hour was the first, and the duration of the effect was the least in the children.

TABLE No. V.

B. Dinner, 12½ p.m., with the Amount and Hour of greatest increase of Pulsation.

	Æt. 6 years.		Æt. 8 years.		Æt. 33 years.		Æt. 36 years.		Æt. 39 years.	
	Pulsa- tion.	Hour.	Pulsa- tion.	Hour.	Pulsa- tion.	Hour.	Pulsa- tion.	Hour.	Pulsa- tion.	Hour.
1st, 2d, and 3d day . . }	14, 6, 8	2, 2, 3	20, 17, 16	2, 2, 3	9, 8, 15	2, 2, 2	18, 15	4, 2	15, 5, 14	3, 1, 2

The averages were, 9·3, 17·6, 10·6, 11·5, 11·3 = 12 medium. Thus, except in æt. 8, the effect was less than that of breakfast, as 12 to 15. The variations were great, and the maximum hour was at least one hour later than that of breakfast. The effect is thus more tardy and sustained, and especially at middle life.

TABLE No. VI.

c. Tea, 5½ p.m., with the Amount and Hour of greatest increase of Pulsation.

	Æt. 6 years.		Æt. 8 years.		Æt. 33 years.		Æt. 36 years.		Æt. 39 years.	
	Pulsa- tion.	Hour.	Pulsa- tion.	Hour.	Pulsa- tion.	Hour.	Pulsa- tion.	Hour.	Pulsa- tion.	Hour.
1st, 2d, and 3d day . . }	0, 9, 8	0, 2, 1	1, 10, 14	1, 1, 2	7, 12, 0	2, 2, 0	8, 6, 6	2, 2, 2	2, 4, 3	3, 1, 1

We have now arrived at a meal which does not always increase pulsation, and thus the averages become less valuable. The effect was much less than that of breakfast and dinner. Thus the averages were, 5·6, 8·3, 6·3, 7·3, and 3 = 6·1 medium; about one half that of dinner, and two

fifths that of breakfast. The variations were very great in the same and in different persons.

During the examinations I took a hearty tea, with bread and butter, after 11 p.m., and sometimes after 4 a.m. This effect may therefore be isolated from any other agency, and was as follows, at the next examination :—6, 5 (6 and 4 at the subsequent examination = 10), 4 = $6\frac{1}{4}$ medium, a number corresponding with the foregoing results. The increase of respiration was, $\frac{1}{2}$, 2, $4\frac{1}{2}$, 1 = 2 medium. When a glass of wine was taken (instead of tea), no effect was evident on the pulse at the next examination, but the respiration was half a respiration quicker.

TABLE NO. VII.

D. Supper, 8½ p.m., with the Amount and Hour of greatest increase of Pulsation.

	Æt. 6 years.		Æt. 8 years.		Æt. 33 years.		Æt. 36 years.		Æt. 39 years.	
	Pulsa- tion.	Hour.	Pulsa- tion.	Hour.	Pulsa- tion.	Hour.	Pulsa- tion.	Hour.	Pulsa- tion.	Hour.
1st and 2d } day . . . }	17, 0	3	15, 9	2, 3	3, 0	3	6, 0	1	0, 10	0, 2

Two suppers only were taken by each person during the examinations, and in one half of the observations there was no increase. The effect is too uncertain to be fairly estimated ; but it is much less on the average (not in individual returns) than any other meal.

Thus, upon a review of the whole, the most effective meals were breakfast and dinner, and then tea and supper in their order, and the effect was, 15, 12, 6, and ? pulsations respectively. The breakfast had also the earliest effect. The latest effect was noticed in the oldest persons chiefly.

RESPIRATION.

TABLE No. VIII.

The following Table represents the Amount and Hour of increased Respiration after every meal on each of the three days.

	Æt. 6 years.		Æt. 8 years.		Æt. 33 years.		Æt. 36 years.		Æt. 39 years.	
	Respiration.	Hour.	Respiration.	Hour.	Respiration.	Hour.	Respiration.	Hour.	Respiration.	Hour.
1st, 2d, and 3d day.										
Breakfast . .	3½, 6, 6½	1, 1, 1	5½, 1, 4	3, 1, 2	3½, 7, 7½	3, 1, 3	4½, 3½, 5	1, 2, 1	2½, 3, 3½	2, 2, 1
Dinner . . .	4, 4½, 5	1, 2, 2	0, 0, 2½	0, 0, 2	3, 3, 2	3, 1, 3	0, ½, 1	0, 1, 1	2, 2½, 2½	2, 3, 3
Tea	3, 0, 4	1, 0, 3	0, 4½, 0	0, 1, 0	4½, 3, 4	2, 1, 2	½, 2, 0	1, 1	½, 3, 3	1, 1, 2
1st and 2d day										
Supper . .	0, 0		0, 0		0, 0		0, 2	0, ½	0, 0	

The averages were, breakfast 4·4, dinner 2·1, tea 2·1.

This table has exceeding interest. Thus, in 16 out of 55 meals there was no increase, and, at supper, only once in ten times was there any increase; whilst at breakfast the increase was amazing, so much as more than one third of the total respirations before that meal, in several instances. In every case, and at nearly every breakfast, the increase was very great, and there was no breakfast without an increase. The influence of dinner was much less, and was null on three occasions; and in both respects closely paralleled the tea meal. The relative effects upon respiration in the five cases, of all the meals combined, may be represented by the following succession of numbers, the cases being arranged in order: (æt. 33) 37, (æt. 6) 33, (æt. 39) 22½, (æt. 36) 19, and (æt. 8) 17½. Thus it is not in the order of age.

Duration of the increased Pulsation and Respiration.

The chief effect is commonly produced within one hour after commencing a meal, sometimes it is the second (but not

later), and after that it declines before the next meal; but with frequent meals the pulse does not always fall before successive meals to the point at which it was found before the first meal. Hence its full influence may not be seen, and therefore the true duration is not always of easy attainment.

It varied from 1 to 4 examinations, but was most frequently 3 examinations. It was less at breakfast, tea, and supper, than at dinner, and also in the children, in whom also the effect was commonly greater and more speedy. The greatest uniformity in all the meals was in æt. 8 and æt. 33, and the greatest disproportion between the meals in æt. 36. It is thus evident that meals have two general effects: 1st, to increase pulsation and respiration; and 2d, to sustain that increase; and as they differ in both, some kinds of food may be most fitted to produce one, and other kinds the other effect. The dinner had the most enduring influence, and it consisted more of animal food, and probably of a larger quantity of solids. Thus quantity as well as quality may be fitted to cause a more enduring increase.

Respiration obeyed the above rules for pulsation.

Prolonged Fasting; its effect upon the Pulse and Respiration.

On October 28th, the children took a good supper at 7½ p.m., and the adults at 8½ p.m.; and no food was again taken until 2½ p.m. on the next day by the children, and 6½ p.m. by the adults. The children took a little water on rising, at 8 a.m. No marked want or distress was felt, except at the usual hour of breakfast; but all were fatigued from the repetition of the observations. The pulse and respirations were noted in the three postures of lying, sitting, and standing, from 8 a.m. to 8 p.m., inclusive; but the returns in the lying posture are alone needed for comparison with those preceding. These have been added, as dotted lines, to Diagram No. 2.

The universal and marked effect was to lower the pulsations, and to keep them low with some uniformity, as is

well shown when the dotted line of lying is contrasted with the dotted line indicating the average elevation with food.

Two circumstances opposed this. First, there is a limit below which the pulse does not fall, and that was attained so early as 9 a.m. by æt. 39, 10 a.m. by æt. 33, and 1 p.m. in æt. 36. Second, there was a manifest disposition in the pulse to rise at the customary hours of its rising, although in the absence of any usual cause for that rise. This was at the usual meal hours, as seen in every case. This led to two ascents in every case, at the following hours.

TABLE NO. IX.

Æt. 6 years.		Æt. 8 years.		Æt. 33 years.		Æt. 36 years.		Æt. 39 years.	
1st ascent.	2d ascent.	1st ascent.	2d ascent.	1st ascent.	2d ascent.	1st ascent.	2d ascent.	1st ascent.	2d ascent.
a.m. 9 to 10	p.m. 1 to 2	a.m. 10 to 11	p.m. 12 to 2	a.m. 10 to 11	p.m. 12 to 3	a.m. 10 to 11	p.m. 3 to 4	a.m. 9 to 10	p.m. 1 to 3

The hours thus corresponded much in each case, and the elevation was the greatest in the children, in one of whom it was 14 pulsations.

There were three corresponding descents: 1st, invariable and very great, at 9 a.m., when the pulse formerly rose greatly from the breakfast; 2d, after the first elevation; and 3d, in the adults only (as the children had then taken food), after the second elevation. This is of great interest, and is probably due to "habit." It is involuntary, and not even connected with the sentiment of expectation (so to speak), for the pulse fell, and did not rise at the morning meal, when the system would be "expecting" the supply. It is also probably due to the protective principle of reaction, and thus aids to defer injury or death in starvation. It is so also in phthisis.

It should be mentioned, in connection with the elevations referred to, that when food is taken at regular hours, there

is sometimes a rise of pulsation (not of respiration) at the hour before the meal, and chiefly at 12 and 4 p.m. This is shown in Diagram No. 1, and to the greatest extent in the most sensitive child, æt. 8.

The measure of the results from fasting may be made by comparing those results with the average results when food was taken; and both are shown on Diagram No. 2. Thus both functions were lower in all cases, and to an average extent varying in pulsation from 8·8 to 13·8, and in respiration from 0·4 to 3 per minute during the hours indicated. The total average showed a subsidence of 10 pulsations and $1\frac{1}{3}$ respiration. It was the largest in æt. 33, and the least in æt. 39; but in all it was mainly due to the low range induced at the breakfast hour; and from other investigations I am induced to regard the breakfast meal as so important in reference to the future pulsation of the day, that I believe a much higher rate would be attained during a prolonged fast commencing after the breakfast, than one of equal duration beginning from the supper.

I have ascertained that in some cases of phthisis the effect of a short fast was to increase slightly the total pulsations of the day. After the above-mentioned fast, in health, the effect of food was increased, and by continuing the examinations five hours and a half after the children's dinner, and an hour and a half after the adults' dinner, the total average was so greatly increased, that during the thirteen hours (8 a.m. to 8 p.m.) the average pulsations were, in the children, nearly the same without as with food. This power of compensation tends to produce a somewhat uniform rate of pulsation under varying circumstances. The examination terminated too early to enable the same results to be obtained in the adults.

The effect of fasting upon the respiration was much less than upon pulsation, except in æt. 36. It was commonly to lessen that function, but there is a want of uniformity in the results obtained. Thus, during fasting, there was a high rate of respiration as compared with the pulsation.

The average ratio of respiration to pulsation during fasting

was greater than with food, in every case except æt. 39, and in that it was less.

2. *Immediate effect of Food.*—An effect upon the pulse and respiration is produced whilst masticating the first mouthful, and I have noted it to amount to 8 pulsations per minute in two minutes, in an adult, and to 23 pulsations per minute in three minutes, in a child.

The following table represents the influence of the breakfast at intervals of five minutes, on October 25th; the persons occupying, and the examinations being made, in the sitting posture.

TABLE No. X.

*Increase of Pulsation from Breakfast, 8 $\frac{1}{2}$ p.m. Sitting posture.
Oct. 25th.*

	Æt. 6 years.	Æt. 8 years.	Æt. 33 years.	Æt. 36 years.	Æt. 39 years.
In 5 minutes	7 E*	4 E	20 E	10 E	8 E
10 "	31 E	8 E	20 E	13 E	16 E
15 "	17	8	20 E	4 E	20 E
20 "	31 E	12 E	20 E	7 E	18 E
25 "	27	14	16	17 E	20 E
30 "	19	26	16	15	10
35 "	31 E	12	12	10	8
40 "	27	14	13	11	4
45 "	17		12	13	6
50 "	23	12	10	9	3

* The letter E signifies the period of eating.

Thus, in five minutes the increase varied from 4 to 20 pulsations; but, except in the latter instance (æt. 33), that was not the maximum obtained from the meal. In ten minutes the effect was very great (8 to 31 pulsations), and was then the maximum in two cases, æt. 6 and æt. 33. In fifteen minutes another maximum was added (æt. 39). In twenty-five minutes a fourth maximum, and, in thirty minutes, the fifth maximum. In æt. 8, æt. 36, and æt. 39,

the pulse progressed to the maximum, and then declined; whilst æt. 33 obtained its maximum at once, and after a time declined. Æt. 6 had the peculiarity of a repetition of the maximum three times, with alternate subsidences of the pulse. Thus the full effect may be obtained, even in five minutes, and the increase from the breakfast may reach 31 pulsations, with a pulsation of 120 per minute. It was the greatest in the children. In three quarters of an hour it was, on the average, as low as it had been in five minutes.

The next table exhibits a similar inquiry at the dinner of that and the following day.

TABLE NO. XI.

Increase of Pulsation from Dinner. Sitting posture.

	October 25th, 1 p.m.		October 26th, 5 p.m.		
	Æt. 6 years.	Æt. 8 years.	Æt. 33 years.	Æt. 36 years.	Æt. 39 years.
In 5 minutes	12 E*	25 E	11	14	8
10 "	11 E	7	23	18	16
15 "	18 E	37 E	15	10	16
20 "	15 E	35 E	7	16	16
25 "	4	21	7	8	26
30 "			17	7	18

* The letter E signifies the period of eating, but in the three last columns that fact was not recorded.

The highest pulsation was again in the children, and was 118 and 112. The effect, in five minutes, was greater than at the breakfast, viz., 25 per minute in æt. 8 (except in æt. 33, and it was equal in æt. 39), and the greatest increase was 18, 37, 23, 18, and 26, a number, on the average, greater than from the breakfast. The maxima were attained twice in ten minutes, twice in fifteen minutes, and once in twenty-five minutes, and were thus a little earlier than after the breakfast. The increase was not proportionate at the two meals in the children; but it was so in the adults.

The effect is much greater after a prolonged fast, as is proved by the returns of the dinner after the fast of eighteen hours and a half on October 29th. On that occasion, in five minutes only the increase was so much as 28 and 26 pulsations, and in fifteen minutes this was increased to 42 and 32 pulsations. The maximum in æt. 6 (the only one who greatly enjoyed the dinner) was much more than twice as much as without fasting, but, in æt. 8, it was a little less than with food. Thus the greatest increase from food may be in the afternoon. The immediate effect of food may thus be so large as to produce an increase of 28 pulsations in five minutes, and 42 in fifteen minutes; but it varies as much as the appetite of persons, or of the same person, at different meals or on different days. The precise influence of dinner and breakfast, in the sitting posture, was, on the average, 24·4 and 22·8 pulsations respectively.

To what direct agency is this sudden and great increase of pulsation due? It is due, probably, in great part, to interference with respiration during the acts of mastication and deglutition. This I have ascertained by careful experiment. Whilst eating simply dry toast and butter, and before the food left the mouth, the pulse rose 6 pulsations, and then subsided partly on ceasing to masticate, and was renewed on the renewal of the process. In Table No. 3, æt. 6 has alternations of elevated and lessened pulsation, viz., 120 followed in five minutes by 106; again, 120 followed by 116 and 110; and again, 120 followed by 116 and 106. The larger pulsation was at the moment of eating, and the lesser when each course was finished. Again, at dinner, æt. 8 had a similar alternation, for the same reason, viz., 100, 82, 112, 100, and 90 when she had finished. Also at the dinner, on October 29th, æt. 8 had the following pulsations at intervals of five minutes, 116 whilst eating meat, 104 when nearly ceased, 122 with the pudding, 118 when ceased to eat. The same case had further an increase of 23 pulsations in three and also in five minutes, whilst eating, at tea, on November 9th, but of 90 in five minutes after-

wards, when she had ceased to eat. When the act was continuous there was no intermission of pulsation.

The explanation is this. During mastication and deglutition the breathing is interrupted, laboured, and very variable in rate and depth. This is more so during rapid and voracious eating. In the act of drinking, even, a deep breath is taken whilst drawing the fluid into the mouth, and is then retained until the fluid has been swallowed, when a rapid expiration occurs, followed by quick respirations or gasping. This is an evident and great interference with respiration. So in the mastication of solid food: the breath is first taken deeply, then expelled inefficiently, so that the respiration is retarded, or catching, during the act, and after swallowing the respirations are more rapid for a moment. As during any meal these acts of mastication and deglutition are often repeated, so is the interference with respiration increased; but it is evident that the more quiet and deliberate is the act, and the smaller the morsels, the less (within limits) will be the interference.

Whenever, without these acts, the respiration is disturbed, the pulse is for the moment slower, and then urged on as if in jets (the effect being proportionate to the violence of the cause). This is well seen in yawning in sensitive children, in shouting, crying, sighing, or other temporary violent respiratory acts. Hence the interference with respiration in eating will, with each act, cause a temporary increase of pulsation, and the repetitions of the act maintain and add to (within limits) this increase. The effect is due both to interference with frequency and depth of breathing.

I am reluctant to attempt any general deductions, or even inferences, from the foregoing inquiry, lest, if in the opinion of any, those inferences should not be borne out by it, the reputation for truthfulness of the inquiry itself should be injured. The two subjects are, however, quite distinct; and the inquiry itself may be sound and truthful, whilst the inferences may be questionable. The former results I offer as facts, the latter as opinions.

Inferences.

The respiration is a most varying function, and especially in children, as is proved by the process of counting; and inferred from its subordination to vocal efforts, bodily exertion, emotions, and eating. The rate of pulsation, in like manner, varies greatly; but the ratio of the two functions is rather influenced by respiration.

Whilst the varying ratio of respiration to pulsation implies that neither function is necessarily dependent for its rate upon the other, it is evident that the circulation is greatly controlled by the respiration, and especially in children and sensitive people. This is proved by the immediate effect of yawning, sighing, shouting, and other momentary emotional respiratory efforts; and by the effect of mastication and deglutition.

The young have less firmness of resistance to adverse influences, as is proved by the facts that the ratio of the two functions is the least in them, and is at the same time the most variable.

Vital actions are at their maximum in the day, and their minimum in the night, and the action of the one and the negation of the other may be in excess or defect, and need regulation. Thus the action of the day may be in excess, from great sunlight, and from too much or too frequently repeated food; or too variable by long intervals between meals; whilst the negation of the night would be too great if the food taken in the day had been in defect, or the daylight had been absent. So the former may be in defect, from the absence of light, food, and wakefulness; and the latter in excess, from the presence of those influences. In various states of the system food should be withheld in the day; and in others, or even in the same, given at night.

So it is evident, from the negation of the night, that night labour, whether it be study, bodily work, travelling, or marching, must be performed when the system is the least able to bear it, and, consequently, when it will be less efficiently performed, or performed with more wear and tear of the system.

Sleep is accompanied by a much lower state of both functions, in the night, than can be obtained by sleep during the day. Hence day sleep is less beneficial than night sleep, and, as the lowest state of these functions is not attained until the second or third hour of sleep, early retiring to rest obtains better sleep than rest sought in the early morning, when the rate of both functions is increasing.

As the pulse and respiration will fall in the evening, and rise in the morning, under all circumstances of health "early to bed and early to rise" must be a natural injunction.

The system becomes charged with material as the meals are repeated during the day, as is shown by the fact that the pulse did not fall after any meal (with the meals taken at 8 $\frac{1}{2}$, 12 $\frac{1}{2}$ a.m., 5 $\frac{1}{2}$ and 8 $\frac{1}{2}$ p.m.) so low as before the meal, and especially as before the first meal. Hence, with meals so frequent, nature calls for a less quantity, and a less stimulating and enduring quality of food, in the latter part of the day; and the effect upon the functions is less considerable and enduring. If no food be taken until the evening, the effect of that meal is inordinate, which would not be the case if the lessened effect of the evening meal resulted from lessened nervous power.

The absence of food in the night, and the greater effect of breakfast, coupled with the rapidity with which that effect passes away, imply that the breakfast should be a good meal, and should be followed by another within three or four hours. The duration of the effect of the dinner is due to the remaining influence of the preceding meal, as well as

to the quality of the dinner food, and implies that considerable time should be allowed to elapse before another meal.

In medical practice we may decrease pulsation by darkness, day fasting, and the lying posture; and then food may be given in the evening, night, or early morning; and increase it by light, food, and the standing or sitting postures. An absolutely even rate is not attainable; but a low and tolerably uniform rate results from long, unbroken fasting, and from night; and a high, and tolerably uniform rate, from frequent, moderate meals, leisurely eaten. Animal food not only increases, but maintains for a longer period, the rate of both functions. The rate of both functions, from 9 a.m. to 9 p.m., may be regarded as tolerably uniform, if food be taken frequently and very moderately; but there is a great difference between the day and the night rate. A rising, or a rapid rate, after 9 p.m., is unnatural; but an increasing rate after 3, 4, or 5, a.m., is natural.

What is the reason for the low ratio of the two functions in the young?

**EXPLANATION OF THE DIAGRAMS ACCOMPANYING
THE PAPER ON THE HOURLY RATE OF PULSATION
AND RESPIRATION IN HEALTH.**

No. 1, exhibits the rate of both functions in the lying posture, at each of 72 successive hours, or three days and nights. It is divided horizontally into three portions. The upper portion has reference to two female children, *set.* 6 and 8; the middle portion to two adults (females), *set.* 33 and 39; and the lower to one adult (male), the observer, *set.* 36. In each portion the upper lines refer to pulsation, and the lower to respiration. The three vertical double lines represent the hour of midnight, and the intervening spaces are each divided into twenty-four parts, each representing an hour. The vertical thin lines show the hours at which the meals were taken. The shaded part represents the hours of darkness, and the part left white the hours of sunlight. The letter S signifies that the subject of examination was then asleep, whilst $\frac{1}{2}$ S signifies a state of half-asleep, or a state which appeared to the observer to be neither asleep nor wakefulness. There are also brackets at the top of the diagrams, showing the hours during which the examined were up, and in bed. The amount of the rate is indicated by transverse lines, and a scale on either side of the diagram.

No. 2, represents the same facts as the same persons on an average, derived from the returns of the 72 hours. Each figure is devoted to a case, and is constructed on the fashion of a clock-dial, with twenty-four radii, to represent the twenty-four hours.

On the outer circle is inscribed the hour, and at intervals, the period of meals. The outer boundary line of the figure represents the pulsations; the inner one represents respirations at each hour; whilst the dotted lines in a part of each figure show the rate of pulsation and respiration during a long fast, as opposed to the ordinary average rate when food was taken. The scale is attached to the concentric circles.

The shading occupies the hours of darkness at that season of the year (November), and the white part represents the presence of sunlight.

ON
ATROPHY OF THE BRAIN,
WITH
CASES IN WHICH THERE WERE REMARKABLE INEQUALITIES
OF THE CEREBRAL HEMISPHERES,
ATTENDED WITH
HEMIPLEGIA AND CONTRACTION OF THE LIMBS ON THE SIDE
OPPOSITE THE ATROPHIED HEMISPHERE.
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THE occasional occurrence of atrophy of the brain was known to the ancients. *Liebanti* supposed it to happen from old age. *Morgagni*¹ refers to cases in which the side opposite to that injured by wounds or blows was paralysed, and convulsive motions occurred in either one or the other side, and sometimes in both. *Valsalva*² found paralysis on the side of the body opposite to the injury of the brain. *Daniel Hoffman*³ observed, in a boy who had suffered concussion of the brain on the left side, with “a

¹ ‘Morgagni,’ let. li, art. 10.

² art. 45.

³ art. 48.

considerable loss of substance, a very violent convulsive agitation of the left foot, and paralysis of the right side, which made it altogether immovable." *Meckel*¹ states that atrophy of the nervous system may be primitive, as in "tabes dorsalis," or consecutive, as in wasting of the optic nerve, when the eyesight is destroyed: he further states, that children of successive births are subjected to similar malformations, and instances three brothers and sisters with deficiency of brain. From *Otto*² we learn that not only in many idiots is the whole brain remarkably small, but that it is generally, though more frequently only locally, very much diminished by external pressure. *Greding* observes, that not unfrequently in the insane the two halves of the brain are formed unsymmetrically³. Atrophy of a portion of the brain is almost always accompanied by imperfect volition, the person being partly deprived of the use of a limb on one side of the body. Many have, in a greater or less degree, muscular contraction of the paralysed limbs. In general the cerebral lesion is on the opposite side to that paralysed, and the individuals are infirm; many are epileptics, and the paralysed limbs are wasted and shortened. *Cazanvieuilh* found, in all the cases which he investigated, except one, the atrophy of brain and paralysis of limbs were on opposite sides, and in the only exceptional case there was a tumour on the contrary side to that wasted. He also observed that the bones of the paralysed limbs were, in general, shortened,—nine times out of eleven in the legs, and ten out of eleven in the arms, the muscles being also wasted; where the limb was large it was from œdema.

The causes of atrophy of the brain are little known; in some cases the affection is congenital, in others it is preceded by convulsions in infancy. Congenital paralysis depends on an arrest of development, or atrophy of some portion of the encephalon. Atrophy may also be the result of

¹ 'General Anatomy,' Eng. Trans., London, 1837, p. 230.

² *Otto*, translated by South, page 389, et seq.

³ 'Dictionnaire de Médecine,' tome xi, p. 589.

an apoplectic clot, which has produced considerable ravages, and terminated in absorption and cicatrization; and also from the pressure of tumours, or from abscess or softening. In old cases of paralysis, with extravasation after absorption of the coagulum, serous cysts are often found in the brain.

Examples of these several sources and forms of the disease are recorded by *Cruveilhier*, *Andral*, and different authors.

When atrophy takes place before the complete development of the skull, and when the deficiency of brain is not replaced by liquid, the skull is proportionably flattened; on the contrary, if the loss of brain be supplied by serum, the skull may be natural, or even larger than natural, as in some cases of congenital hydrocephalus, where the brain is in a great degree wanting. In some cases the internal table of the skull is unusually thin, and the diploe of great thickness.

Atrophy of the convolutions may be a mere diminution in volume, or a shrivelling of the same, presenting an unequal and granular appearance; this last is often accompanied by different shades of colour, from former effusion of blood.¹ In other cases the irregularities are filled with fluid contained in the subarachnoidal cellular tissue, when circumscribed, in the form of a cyst.

In a paper of mine, published in the 'Edinburgh Medical and Surgical Journal,'² there is an account of 22 cases of cerebral dropsy, which occurred in adults. Of these cases atrophy of the brain was found in 3 males and 4 females; in No. 735 with contracted limbs, bed-sores on hips, ankle, &c.; atrophy of optic nerve in Nos. 746 and 753. In the same journal will be found notes of 17 cases of softening of the brain; paralysis occurred in 10 of these, with contraction of the limbs in 4; in No. 772 there was loss of substance in one cerebral hemisphere, and gangrene of cerebellum, and, in 776, atrophy of the brain, attended with gangrenous sores of the character described by *Cruveilhier*.

The continuation of the paper published in No. 172 of

¹ 'Edinb. Med. and Surg. Journal,' No. 156, p. 159.

² *Ib.*, No. 171, p. 466.

the same journal, Case 787, is an instance of hemiplegia of six years' standing, caused by a tumour on the opposite side. Case 815 affords a similar example.

In 13 cases of paralysis, recorded in the same journal, there was atrophy of the brain in 6, and in 4 the atrophy was observed principally in one cerebral hemisphere.

In 31 cases subsequently examined by me (16 from thirty to sixty, and the remainder upwards of sixty years of age), the brain was below the average size in three fourths of the number, and was unsymmetrical in the remainder. There was cerebral softening in 11 cases; in an acute case, of one week's duration, the softened hemisphere was 3 oz. heavier, and, in another, $1\frac{1}{4}$ oz. heavier than the other hemisphere; in one, there was a loss of $2\frac{1}{4}$ oz., and, in a fourth, the difference was only 1 oz.; there were convulsions in 2; general paralysis in 1; paraplegia and contraction of the lower limbs in 2; hemiplegia of right side, and contracted wrist, in 2, softening being in the left hemispheres; hemiplegia of the left side in 2, and the cerebral softening on the right side; paraplegia and contracted lower limbs in 1, with softening of each "corpus striatum," and sloughing bed-sores. In one, loss of substance in left cerebral hemisphere, and hemiplegia of right side. Apoplexy existed in 2 cases; in one of left corpus striatum and optic thalamus, with hemiplegia on same side; the other a rusty colour, and wasting of cerebral convolutions of left hemisphere, and hemiplegia of right side. In 1, tumours, and considerable atrophy of the brain, which weighed only $27\frac{1}{2}$ oz.; the lower limbs were contracted. Fluid was found in the ventricles, or on the surface of the brain, in 11 cases; in 1 there was a difference between the cerebral hemispheres of 2 oz.; in 8, paralysis, with more or less wasting; in 2 there was stupor, with no paralysis; and bed-sores in 3. Atrophy of the brain in 5, wasting of the "pons varolii;" in 1 there was paraplegia of the lower limbs, and the right cerebral hemisphere was 1 oz. heavier than the left; wasting of the right optic thalamus, and a soporific state, in 1; wasting of the optic nerves, in cases of blindness, in 3; one of these, a female, aged 85, was blind of the left eye, and

had atrophy of the left optic nerve; her eyes were dissected by the late Mr. Dalrymple, who found "the sclerotic and choroid perfectly healthy, but the retina was extremely thin and translucent, and, compared with the right eye, had not half its substance; in fact, the granular or external layer appeared nearly absent. The iris had been inflamed, and the pupil was closed upon an opaque capsule, to which it was universally adherent; the lens behind was opaque, the vitreous body healthy. The case had probably been one of iritis originally, which had gone on to closed pupil and spurious cataract in the left eye, but it is singular to find this resulting in atrophy of the retina and optic nerve." There was fluid on the surface of the brain, and atrophy of the cerebral convolutions; there was also $1\frac{1}{2}$ oz. of fluid in the lateral auricles; weight of brain, $36\frac{1}{2}$ oz. Old pulmonary tubercles degenerated into earthy and hardened matter in the apices of both lungs. Heart natural. Abdominal organs small.

There may be considerable loss of substance of the surface of the hemispheres, without hemiplegia, as the following case demonstrates: A female, æt. 55, died of dropsy; enlarged heart ($16\frac{3}{4}$ oz.), left cerebral hemisphere ($3\frac{1}{4}$ oz.) smaller than the right, the convolutions of a yellow colour, and softened to the depth of one fourth of an inch; there was no paralysis, but there was a deficiency of intelligence. Weight of brain, $41\frac{1}{2}$ oz.

A case in which there was a remarkable inequality of the cerebral hemispheres in an intelligent man, attended with hemiplegia and contraction of the limbs on the side opposite the atrophied hemisphere, which was only half the size of the other, was published in a contribution of mine to the 'Edinburgh Med. and Surg. Journal,' No. 172, Case 823. The cerebral convolutions were wanting on the left side, and their place was occupied by cellular membrane, filled with transparent fluid; the medullary portion of hemisphere was unusually firm. The weight of brain was $33\frac{1}{4}$ oz.; the thoracic and abdominal organs were natural; death occurred from convulsions, and was sudden. This case would seem to

be favorable to the hypothesis of the late Dr. Wigan on "the duality of the mind," as it appeared, from the evidence of his sister, that this man possessed a more than ordinary amount of ability, being a good accountant.

A case somewhat similar to the foregoing, but of the history of which fewer particulars could be obtained, occurred in a messenger, æt. 30 years, who died of pulmonary phthisia. He had hemiplegia and contraction of left wrist and ankle for twenty-eight years. The dura mater was quite flaccid over the right cerebral hemisphere, which was not half the size of the left; the convolutions were firm, but much shrivelled; the pia mater in an cedematous state; 1 oz. of fluid in lateral ventricles; the right cerebral hemisphere weighed 9, the left $19\frac{1}{2}$ oz.; weight of brain, $83\frac{3}{4}$ oz.

An epileptic male, aged 18, suffered from epilepsy since his fifth year. He was found dead in bed. Hemiplegia of the right side; right wrist and ankle firmly contracted; right lower extremity one inch shorter than the left; right thigh two inches less in circumference, and calf of right leg two inches and a half less in circumference than the left. The circumference of right arm and forearm two inches less than left. There was an extensive loss of substance laterally of the anterior lobe of the left cerebral hemisphere; the space filled by fluid contained in a reticular membrane; the cerebral structure beneath the membrane smooth, unusually firm, and of a brown colour.

Of the cases now referred to 33 were males, and 31 females. One male only was below 30 years of age; and of the other males, one half were between 30 and 60 years, and the remainder above that age, the oldest being 84. The youngest female was 32; 12 were under 60, 18 above that age, and the oldest 98.

The average weight of the brain in the males was $43\frac{1}{2}$, in the females $39\frac{1}{4}$ oz. The smallest brain in the males was $30\frac{1}{2}$, in the females $27\frac{1}{4}$; the largest in the males was $52\frac{1}{2}$, and in the females $50\frac{1}{4}$ oz. No account has been here taken of any case where the difference in weight between the cerebral hemispheres was less than one ounce.

As regards the relative frequency of atrophy of the brain (with inequality), it was found in about $4\frac{1}{2}$ per cent. of the adult cases examined by me at the St. Marylebone Infirmary. According to my experience subsequently, it is at least twice as frequent in the insane, and males of this class are very much more subject to it than females; and it is also very common in epileptics.

In 31 cases in the insane (21 males and 10 females), a difference of one ounce between the cerebral hemispheres was found in 7 males and 4 females. Of these 2 males and 1 female had general paralysis; and in 1 female, æt. 61, who always sat bent forward, and tottered in walking, there were several semicartilaginous specks of various sizes, from that of a split pea to a silver fourpenny piece, on the arachnoid of the spinal cord, the largest over the "cauda equina." The specks are indicated in the drawing sent with this paper. There was also a mass of hydatids on the under surface of the right lobe of the liver. In 1 male the difference was $1\frac{1}{4}$ oz., and the case one of general paralysis; the brain unusually small, 39 oz. In 5 males and 4 females the difference was $1\frac{1}{4}$ oz.; in 3 of the males and in 1 female there was general paralysis, and in 2 females cerebral softening. In 2 males and 1 female the difference was $1\frac{1}{2}$ oz.; in both males there was general paralysis, and in the female epilepsy. In 1 male the difference was 2 oz., and in this case there was general paralysis. In 1 male, a case of epilepsy, a difference of $2\frac{1}{4}$ oz. In 1 male and 1 female the difference was $2\frac{1}{4}$ oz.; the man died in convulsions, and the woman had epilepsy. In 1 male the difference was 3 oz., a case of epilepsy. In another male epileptic case, the difference was 4 oz. In 1 male the difference was 6 oz.; there was hemiplegia and contraction of the joints upon the opposite side; the right cerebral hemisphere was destroyed to the depth of an inch, and three inches and a half in the antero-posterior, and two inches and a half in the lateral diameter; skull natural. The symptoms were those of mania, combined with epilepsy. In one male the cerebral hemispheres were equal in weight; there was blindness of

left eye for twenty-two years (caused by an accident from lime), and the left optic nerve was only half the size of the other; this was a case of congenital idiocy.

The average weight of the brain in the 21 insane males was $45\frac{1}{2}$, and in the 10 insane females $41\frac{1}{4}$ oz. In 10 males and 4 females the atrophy was of the right cerebral hemisphere; and in 9 males and 5 females of the left. In one male and one female the atrophy was general; and in one male local, being confined to the optic nerve.

Atrophy of the Brain in Insane Persons.

CASE 1.—A female, H. R—, æt. 24, was brought to this asylum paralysed, and in a very bad state of bodily health; she lived only eight days after admission.

It was stated that she met with an injury to the head thirteen years ago, when she and two other persons were thrown out, and one killed, by the upsetting of a cart coming rapidly down a hill. She was never fit to follow any occupation afterwards.

The head was of the natural size; the skull was unusually thick, the brain much wasted, the dura mater quite flaccid, a gelatinous fluid in the pia mater, the lateral ventricles enlarged and filled with clear fluid, the structure of the brain firm; each cerebral hemisphere weighed only 13, and the entire brain 31 oz. The membranes of the spinal cord were red, and the vessels turgid with blood.

CASE 2.—A German, a teacher of languages, æt. 27, admitted in a state of incoherence, with some symptoms of general paralysis; occasionally very violent; seized with spasm and rigidity of the muscles of the back nine days before death; appetite, which had been excessive, fell off suddenly. Head large; the dura mater, over right cerebral hemisphere, distended; and, when divided, there was nearly 8 oz. of fluid, of a dark colour, mixed with blood, and contained in a smooth, membranous sac, within the cavity of the arachnoid; the surface of that hemisphere was smooth, and much

smaller than the other. The brain, in other respects, appeared natural. The right cerebral hemisphere weighed $12\frac{1}{2}$, and the left 17,—the entire brain 36 oz. There was an unusually large quantity of fluid in the spinal canal, and about one inch of the lower end of the spinal cord was softer than natural; pleuro-pneumonia on both sides of the chest. Although in this case there was a difference of $4\frac{1}{2}$ oz. in the cerebral hemispheres (the loss of substance being from the surface), there was but slight paralysis, and neither convulsions nor epilepsy: duration of insanity, eight or nine months. In another case before mentioned, of twenty-eight years' duration, where the loss of substance in one cerebral hemisphere amounted to 6 oz., there was hemiplegia and contraction of the joints on the side opposite; and also epilepsy, reported to have been caused by an injury to the head. He lived to the age of 47 years.

Atrophy of the Brain in Epileptics.

Amongst 32 male and 33 female epileptics at present in this institution, there are 3 strongly marked cases of hemiplegia and contraction of the limbs; and in 1 there is a manifest depression of the cranium on the side opposite the paralysed one.

. CASE 1.—W. M—, a male, æt. 28, when brought to this asylum three years and a half ago, was reported as having, from infancy, been subject to epileptic fits, which were of frequent occurrence. His parents were agriculturists; he had received no education; had no occupation; he is cleanly in his habits; his mental state one *approaching* idiocy; his expression is characteristic of the state of his mind; his disposition cheerful; his bodily health indifferent; his pulse 78, not so frequent as in other epileptics; height, 5 feet $3\frac{1}{2}$ in.: weight, 133 lb. Has hemiplegia of the left side; the elbow and wrist contracted; leg shortened; right arm 2 inches longer, and $1\frac{1}{2}$ inch larger in circumference than the left;

the right leg $1\frac{1}{2}$ inch longer, and three fourths of an inch more in circumference than the left ; the head is small, with a retreating forehead, narrowing at the top ; the circumference $20\frac{1}{4}$ inches, measured round the middle of the forehead and occipital protuberance ; $12\frac{1}{2}$ inches from root of nose to occipital protuberance, the antero-posterior measurement ; and $12\frac{1}{2}$ inches from centre of one external auditory foramen to the other, transverse measurement. In this case there is atrophy of the right cerebral hemisphere, arising probably from arrest of development.

[This patient has, since the paper was read to the Society, died of lumbar abscess not connected with any disease of the spine. He was under medical treatment, and confined to bed for the last ten weeks with diarrhoea and hectic fever, and had an ulcer on the coccyx, and another on the hip ; he became much emaciated, and lost 40 lb. in weight during his illness.

Head—skull thick, unequally so ; the lower surface shows the sinuses dilated, and skull thickest on the right side, being the corresponding side to the small cerebral hemisphere ; the dura mater thickened and flaccid, especially over the anterior portion of the same hemisphere ; the falx imperfect, wanting at the anterior part. The cerebral convolutions smaller on the anterior lobe of the right than of the left hemisphere, and the left hemisphere anteriorly encroached on the right. The brain very small, being only 30 oz. avoirdupois in weight, one pound below the average in the male. Chest—old pleuritic adhesions on the right side, and the lower lobe of right lung in a softened state from low pneumonia, weight 18 oz. ; left lung natural weight, 8 oz. ; heart filled with dark coagula, weight, $8\frac{3}{4}$ oz. Abdomen—stomach large, weight 6 oz. ; liver simply enlarged, but in an excessive degree, weight 74 oz. ; spleen dark coloured, weight $6\frac{3}{4}$ oz. ; pancreas, 3 oz. ; right kidney, 6 oz. ; left kidney, $5\frac{1}{2}$ oz. ; on the left side, beneath the peritoneum, there was putrid pus found to extend from diaphragm to brim of pelvis, and between fibres of psoas muscle ; no sign of the abscess externally. A drawing of the brain in this case is annexed.]

CASE 2.—A. H—, female, æt. 47, has hemiplegia of left side, left ankle and foot contracted. Is reported to have been healthy as an infant, and no deficiency was observed until she was 7 years of age, and old enough to use her hands; after which the contractions gradually increased, and epileptic fits occurred, which ceased at 10 years of age, but returned in after life, and now occur, with severity, about once a month. Her propensities are to thieve, tell lies, and be abusive, and indecent in her conduct, for which reasons she cannot be kept at home. Her intellect is clear; she can read, and repeat well from memory; no deficiency is observable in the form of the head—circumference $22\frac{1}{2}$ inches, antero-posterior measurement 13, and transverse $13\frac{1}{2}$ inches. Length of right arm $25\frac{1}{2}$, of left arm 24 inches; left hand and forearm shrivelled. The right leg $1\frac{1}{2}$ inch longer, and $\frac{1}{2}$ an inch larger in circumference than the left. In this case the atrophy of the right cerebral hemisphere began in infancy.

CASE 3.—M.A.C—, female, æt. 27, has hemiplegia of right side; wrist, hand, and ankle much contracted and shrivelled; on left side of head there is a manifest lateral indenture, giving proof, in this case, of arrest of development of that side of the brain; she has epileptic fits, and is occasionally very violent and passionate, and will bite even her own flesh. She has a brother who is also an epileptic. Her expression is pleasing and cheerful; there is no great deficiency of intellect, but there is a want of self-control. Circumference of head, 21; antero-posterior measurement, 12; and transverse measurement, 13 inches. Length of right arm 23, left arm $25\frac{1}{2}$ inches; left forearm 1 inch more in circumference than the right.

CASE 4.—E. D—, a female, æt. 19, nearly twelve months in the asylum. Imbecile; disposition cheerful, generally singing; appetite moderate, sleeps well, takes a good deal of exercise. She is subject to fits, which occur night or day, about four or five in a week; they occur without previous

notice, very suddenly, and partake more of the character of eclampsia than epilepsy. Her state appears to depend upon an arrest of development of the right cerebral hemisphere. The limbs on the left side of the body are smaller than the right, and there is a considerable difference between those of the upper and lower extremities, the left being $\frac{1}{4}$ inch shorter than the right, and also $\frac{1}{4}$ inch smaller round the calf of the leg; whilst the left arm is 2 inches shorter than the right, and $\frac{1}{4}$ inch less in circumference. The left wrist is firmly contracted, and the hand wasted, so that it is of very little use.

CASE 5.—J. E—, a female, æt. 29. Mania, combined with hemiplegia, right side, from apoplexy, eight years ago. Right leg and foot each 1 inch shorter than the left, and the calf of the right leg $1\frac{1}{4}$ inch smaller than the left. The right wrist is $\frac{1}{4}$ inch smaller than the left. She is very passionate and violent; not subject to fits. In this case the apoplectic attack would appear to have been destructive to a portion of the left cerebral hemisphere, and have caused the contractions of the limbs on the right side, especially of the lower extremity.

In these five cases the character of the epileptic fits is somewhat similar (the individuals being aware of their approach), and they are neither so frequent nor so prolonged as in ordinary cases of epilepsy. In the first case, 18 fits; in the second, 3; and in the third, 13 have only been observed during four months.

Some amount of atrophy of the brain, and irregularity of the cerebral hemispheres without paralysis, is frequent in epileptics; and, in the foregoing cases of general paralysis amongst the insane, a minor degree of inequality of the cerebral hemispheres from softening was also found. The inequality in cases of general paralysis was mostly produced by inflammation, the softened part being usually on the heavier side; and generally, in such cases, the spinal cord was similarly affected—being likewise softened.

OBSERVATIONS
ON
CONGENITAL DEFICIENCY OF THE PALATE,
AND THE
MEANS TO BE USED FOR ITS RELIEF.

BY
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THE frequent occurrence of congenital deficiency of the palate, and its consequent distressing effects upon the voice and articulation—the difficulty of modifying this deficiency by artificially adapted palates, and the inconvenience experienced in their use—the few attempts that have been made, in this country, to remedy the defect by operation, and the want of success that has generally accompanied those attempts—are facts which will, it is hoped, justify me in bringing before the Society a consideration of the means by which the deficiency may be permanently remedied, and the mode of operation by which the deformity may be successfully overcome; especially so, as the following observations are grounded upon the results of cases in which openings in the hard palate have been effectually closed.

The subject of cleft palate has already been brought under the notice of the Society. In 1843, Mr. Fergusson, in a paper published in the volume of the 'Transactions' for that year, pointed out the principles that should guide the surgeon in the performance of the operation for clefts occurring in the soft palate, and little remains to be said on the treatment of the fissure affecting this part alone. I cannot, however, allude to this paper without expressing how much, in my opinion, we are indebted to its author for the improvement he first suggested in the performance of this operation. I allude to the division of the levator palati muscles before attempting to bring the edges of the cleft together by sutures, and thereby ensuring the closure of the soft palate, as far as scientific surgery can ensure the success of any operation.

It will be seen that the observations in this communication are chiefly directed to the consideration of the deformities affecting the hard palate, and the measures which may be applied for their improvement.

Having witnessed satisfactory results in several cases in which the congenital deficiency has been remedied by the union of the soft structures covering the bones, I have thought it right that the profession generally should judge of those results, and decide whether the operations on the hard palate may not rank in importance with those more frequently undertaken by English surgeons. Mr. Fergusson makes the following remarks in alluding to this question: "Until a very recent date no attempt has been made by the surgeon to close the gap in the (hard) palate, excepting by means of an obturator, which has always been supplied by the dentist. Dr. J. M. Warren, of Boston, has, however, closed the fissure here, as well as in the soft parts, by a proceeding strictly surgical. He dissects the soft tissues from the hard vault of the mouth, between the margin of the cleft and the alveoli, and then closes them in the mesial line by a proceeding analogous to the operation in the soft palate, and reports most favorably of the result. I have resorted to the operation in several instances without

success; yet the plan is so clever and reasonable, that I cannot but recommend it for further trial.”¹

There can be no doubt that English surgeons generally have not been alive to the importance of attempting to close the hard palate by operation when congenital deficiency exists, and in this respect we are behind the advances made by several American and Continental practitioners. And, though I have no novelty to relate beyond what is said to have been effected by them in such cases, I hope to establish the fact that the operation may be undertaken with confidence, and that we may reasonably expect a large share of success to reward our efforts. Any minute description of the deformities that affect the palate would be out of place in a communication of a practical character, and would require more time than could be devoted to it on such an occasion as the present. But that the chief deformities might be understood, and as they were referred to occasionally, several models were prepared and exhibited to illustrate their characters. Two preparations illustrating fissure of the palate were also exhibited; one showing the fissure, with the soft parts undisturbed; the other the fissure in the bony palate, the soft parts being entirely removed. These models were all taken from life; and I must beg leave to express my thanks to my friend Mr. Vasey, for the care and trouble he bestowed upon their preparation. They explained clearly, with the natural specimens, the different conditions between the most extensive deformity and that least affecting the hard palate; and which may be thus divided:

1. The first and most extensive variety, extending through the soft and hard palate, and then, dividing in front, passes through the alveolar ridge, making a gap on each side of the incisor teeth.
2. The second extends through the soft and hard palate, and through the alveolar ridge also, by a single gap only, on one side of the incisor teeth.

¹ ‘Practical Surgery,’ p. 613, third Edition.

3. The third passes through the soft and hard palate, and terminates in front immediately behind the alveolar ridge. In this variety there is often great irregularity of the upper incisors.

4. The fourth extends through the soft and about three fourths of the hard palate.

5. The fifth extends through the soft palate and the palate bones only.

6. The last exists as an opening in the hard palate, in which case the soft palate may be entire.

This division may appear unnecessarily minute, but it seems to me a desirable one for several reasons. In the first place, as a general rule, in all cases in which the alveolar ridge is implicated in the fissure, either singly or by a double gap, we have observed that fissure of the upper lip has existed at birth. I may add that the fissure of the upper lip will most frequently be double whenever the fissure in the alveolar ridge is double also. It may be said that this complication will be found to exist as a general rule. I have not myself found an exception occur in all the cases I have examined.

It will be observed, too, that in cases which come under the third variety—that is, where the fissure extends to the alveolar ridge, or rather to the point which indicates the line of union between the analogue of the inter-maxillary with the superior maxillary bones—in these cases fissure of the upper lip will have existed at birth. I have not seen one exception to this remark.

In the second place, the above division of these cases is useful, practically speaking. It may be laid down as a general principle regarding the operations on these parts, that the more extensive the deformity, the more extensive will be the surface of the soft tissues, the greater the facility of bringing the edges of the fissure together, and the more sanguine may the surgeon feel regarding the ultimate success of his operations; whereas in the last two varieties much care is required to close the parts, nor are

the results as frequently successful and satisfactory as in the other instances.

That these remarks may be clearly understood, it is necessary to refer to the model of the most deformed palate, and compare it with the model of a naturally developed mouth. In the greatest extent of deformity, the sides of the fissure are observed to run upwards in a direction almost perpendicular. In a drawing of a sectional view of the same model, the measurement of these sides is marked 2 inches. In a sectional view of a natural palate, the measurement of the arch is $2\frac{1}{30}$ inches. In these two instances there is only the difference of $\frac{1}{30}$ th part of an inch in the measurement of the surfaces of the soft tissue, which, in one instance, covers the sides of the fissure, in the other the arch of the palate. But the measurement of the base of the palate in each case shows a much greater disproportion, being in the natural palate $1\frac{6}{10}$ inch, in the cleft palate $1\frac{4}{10}$ inch; these proportions being in favour of the cleft palate, thus providing an ample surface of tissue, which can be brought together and united in the median line.

If the sides of the fissured palate are separated from the bones, and their edges approximated, as marked by the dotted line in fig. 1, Plate I, they will meet at a point sufficiently high to form an ample arch for the mouth, and even somewhat higher than is necessary, so that generally they overlap each other slightly, and sufficiently to allow a thin strip to be removed on either side.

If the sectional drawing of the Model No. 6 is next examined, it will be seen that though the fissure extends through the soft palate and palate bones, yet the arch of the mouth immediately in front of the fissure has a natural curve. The soft tissues may be freely separated from the bone surrounding the margins of this fissure; but without some other measures are resorted to, there will not be sufficient breadth or freedom in the flaps to allow of their meeting in the median line, nor to allow the sutures to be secured in such a manner that hopes may be entertained of closing the fissure by the union of its edges.

Thus it will be found that the lesser the fissure in the bony palate, the more natural the curve of the arch, a condition which adds to, rather than diminishes, the difficulties of the operation.

These points will, I trust, sufficiently explain the objects to be obtained in the treatment of these cases. I shall endeavour to describe the different steps of the operation by the account of a few cases in which it has been attempted.

A gentleman, æt. 24, affected with cleft of the palate, extending forwards to within half an inch of the incisor teeth, was brought to me by Dr. Maurice Collis, of Dublin. His articulation was very imperfect and unintelligible, and attended with a peculiar cavernous resonance. The soft parts were abundant over the bones, and there was quite sufficient soft palate to give encouragement as to the results of an operation being successful. He consented to the operation, at my suggestion, and it was performed on the 9th July, 1855, Mr. Tatum, Dr. M. Collis, Mr. Pyle, and Mr. Holmes, kindly affording me their assistance.

The edges of the cleft of the hard palate were incised along their whole lengths, with a knife which was made of a flat piece of steel, bent at a right angle about a quarter of an inch from its extremity, and the cutting edge of which was about one eighth of an inch broad. The line of incision ran along the line of union between the mucous membrane of the mouth and that of the nose.

With a much broader knife, in shape like the first, the soft tissues were carefully and slowly detached from the bone, by working the edge of the knife between the attached surfaces, from within towards the alveoli, care being taken to keep the edge of the knife against the surface of the bone. By degrees the flaps were thus sufficiently separated. In front, rather more care was necessary, and a smaller knife, bent at a rather acute angle, was used. A knife with a

blade which is acted upon by a screw, to alter the angle at which it cuts, is most useful in this operation.

At the posterior parts, as there did not appear to be sufficient freedom for the flaps to meet readily, a curved knife was introduced through the palate, near the last molar tooth (on each side), and pushed upwards and inwards between the bone and soft parts, until its point was seen in the gap of the fissure; the blade was then moved slightly backwards and forwards, and on its withdrawal the flaps readily met in the median line. The edges were then pared, and three sutures were introduced. When drawn together, no strain was found upon the flaps, and the parts were accurately adjusted to each other. The covering now formed was for the greater part of the hard palate. The sutures were loosely tied, so that in case of much swelling they should not cut the soft tissues. A good deal of hemorrhage took place occasionally during the operation, but was always readily checked by the pressure of the finger, and, as the patient was sometimes faint, plenty of wine was allowed during the time it occupied. After the operation he was allowed a liberal diet of fluid and pulpy food, but he was not allowed to speak for the first five days.

The first suture was removed on the fourth day, and the last by the end of the week, and he left town a few days afterwards, union being complete.

The cast of the mouth, taken on his return, shows how much palate was gained by the first operation; see also Plate II, fig. 2.

The second operation was performed on the 27th October, Mr. Charles Hawkins, Mr. Pyle, and Mr. Holmes, being present, and kindly assisting me. It consisted in merely bringing the edges of the soft palate together; and although the levator palati muscles were divided, as the steps of the operation differed somewhat from the directions given by Mr. Fergusson, a short account of it is added.

Instead of using forceps to hold the curtains of the divided palate with, sutures were passed through them, and the ends,

being drawn out of the mouth, were tied together on each side, so that either suture could be held in the hand without fear of its slipping out of the flap. The soft tissues could thus be readily put on the stretch, and the object to be obtained by the employment of the forceps was secured in the most efficient manner. All bruising of the mucous membrane was avoided, and a good hold retained of the flap, which enabled it to be drawn easily towards the centre of the gap.

In this case, the flaps of the soft palate were so active and irritable, that it would have been impossible to hold the points of the uvula without exerting much pressure upon them, had the forceps been employed. The flaps being gently stretched, the palato-glossi and pharyngei muscles were divided with scissors.

Whilst the flap was again put on the stretch, a sharp-pointed, double-edged knife was run through the left side of the palate, on the inner side of the hamular process, which can be readily felt through the soft parts. The point of the knife being kept in a direction upwards and inwards, was soon seen to have passed through the soft palate, and to be projecting into the gap of the fissure, above the line of the levator palati. The handle of the knife was next raised, and a sweeping cut made along the posterior surface of the soft palate. It was then withdrawn, leaving but a small opening in the mucous membrane, on the anterior surface of the curtain, and the levator palati was found freely liberated on this side. A similar proceeding on the right side liberated the levator there; but as there was a want of soft tissue, and some strain upon the points of the sutures when the flaps were drawn together, the incision was carried down, on this side, to the free margin of the soft palate. The edges of the flaps were then pared, and found to meet readily in the median line. The operation was soon over, and but little blood lost. The subsequent treatment was the same as on the former occasion. All the sutures were removed by the eighth day, and he left town the following morning.

A small part of the fissure, between the proximate points of the two operations, was purposely left untouched. I was afraid of attempting to unite too much at one time, for fear of sloughing following the attempt, and I have no reason to regret the precaution taken. The parts forming the opening have not been touched on either occasion; its margins have not been pared, nor have its edges been drawn together; so that the present condition of the palate shows what has been effected, and what remains to be done; all that was brought together has united, and there is no fear but that the remainder will do so, when the attempt is made to close the opening. A model was shown illustrating the effects of the second operation.

The next case occurred in the practice of the late Mr. Avery. Having assisted him in all the operations by which this fissure was closed, and having notes of the case in my possession, I have added it to the foregoing one, as an excellent example of a successful result.

A man, æt. 48, had complete cleft of the soft and hard palate, terminating in front, close to the incisor teeth. Unfortunately, a perfect model of the mouth before the operation was not prepared; but an imperfect cast of the upper jaw shows a portion of the fissure.

It was a very formidable-looking gap, and produced considerable imperfection in articulation. The soft parts covering the bones were very thick and velvety, and there was ample soft palate on either side.

The soft palate was first closed in the usual manner; and though the man had a most violent cough, which interfered much with the operation, the greater part of the soft palate united in the median line, but the uvula remained bifid. The sides of the fissure in the hard palate were nearly perpendicular in their direction. He was operated on for this portion of the cleft some few weeks after the union of the soft palate. The plan adopted was much the same as already detailed in the first case; but the knives used were

smaller than those now employed. Lateral incisions were also made, to assist in bringing the edges of the flaps together.

The greater part of the opening closed, but a portion did not unite, and required a subsequent operation. And even after the third attempt, a fissure remained, through which the flattened end of a probe could be passed. The repeated applications of the heated platinum wire to the mucous surfaces eventually obliterated it.

A model of the palate illustrates the present condition of the patient.

I cannot allow the mention of Mr. Avery's name to pass, without expressing how much I was indebted to him for his kindness, in affording me every assistance in the treatment of these cases that his experience could command. Those who witnessed the patience and ingenuity with which he managed operations for cleft palate, will agree in saying that he would have ensured for himself a high reputation had it been permitted that his life should have been spared. Having enjoyed his friendship, and having benefited by his experience, I should not have done justice to my own feelings or to his labours, had I neglected to say thus much of his character, on an occasion when the attention of the Society is invited to a subject in which he took a deep interest, and attained much success.

Another model illustrated another instance of successful closure of the hard and soft palate. This occurred in a young man, in whom the fissure extended through the soft, and the greater portion of the hard palate.

In another model was seen how much could be effected in a case by one operation. And when compared with the conditions previous to operation it showed that the greater portion of the hard palate had been closed.

A model was shown to illustrate how little union may result after an operation. The edges of the soft tissue covering the hard palate were, in this instance, brought

together most satisfactorily, but the patient could not be induced to take sufficient nourishment subsequently, and to this circumstance the failure of the case was partly attributed.

The cases already quoted show that fissure of the hard palate may be closed. It remains to be considered, by what means this alteration is most readily effected.

The first consideration must be paid to the instruments which are necessary in these operations,—a consideration of the utmost importance, as much so indeed as any other point in connection with the treatment of these cases. The general shape of the knives usually employed has already been alluded to ; but as the size of the mouth and the dimensions of the fissure will vary in different individuals, so a variety of instruments, both in size and shape, is indispensable. These varieties will be best appreciated by referring to the drawings and instruments.

The broader cutting edge should be chiefly employed, and in preference to the narrower blades, as it secures greater expedition with less chance of bruising the soft parts. This conclusion was arrived at after witnessing several operations in which the smaller knives were only employed ; and I now prefer, throughout the greater part of the detaching process, the broadest-edged blade that can be conveniently used. I am much indebted to Mr. Blaise, of St. James's Street, for the readiness and skill with which he has carried out my wishes in the construction of several new knives.¹

It is a point of great importance, when cutting between the soft tissue and the bone, to keep the edge of the knife well against the surface of the latter, and gradually, by a half-scraping movement, to detach the flap from it. Any tearing or laceration of the soft parts is generally followed by sloughing ; and the smaller cutting instruments are very apt to run through them, if any degree of force is used, and

¹ The handles of the knives must be strongly connected with the shaft of the cutting edge, as they are required to bear considerable pressure ; so that it is far preferable that the blade and the handle should consist simply of one piece of steel, without any break or joining.

the knife at all slips from the bone—an accident which may occasionally occur, but which will rarely happen with the use of the broad knife.

I believe a point of equally great importance is not to attempt the union of too large a surface at one operation. The attempt to unite the hard and soft palate at once is an extremely injudicious proceeding, and will most likely end in failure.

There are many reasons against the propriety of such an attempt. The mere separation of a large surface of soft tissue from its attachments would materially interfere with the nutrition of the part, and consequently with its union. The anterior and posterior palatine arteries would be divided at one time. I have seen this done, and have seen sloughing supervene upon this division of vessels. The patient too loses much blood by this mode of procedure. The operation must necessarily be a long one, and the patient becomes tired and faint before it can be completed. The positions of the head and mouth, the pain, and the constant washing out of the mouth during the operation, all add to this distress, and cannot be submitted to for long without far greater determination than most patients possess. For all reasons it is much better to be satisfied with closing the fissure by a succession of operations, than to attempt it in one.

In selecting between the hard and the soft palate for the first operation, I prefer commencing with the anterior portion when the fissure extends into the maxillary bones. To close the fissure of the hard palate to a slight extent only, the soft tissues must be freely detached anteriorly, the anterior palatine artery being generally divided. It is not necessary to carry the separation of the soft tissues from the bone as far outwards as the posterior palatine foramina; so that the flaps, though much separated from the bone, derive nutrition in abundance posteriorly, and union takes place satisfactorily.

When firm union has occurred, and the circulation has been fully established in the newly-formed roof and across the median line, it will generally be found that the gap in the soft palate has sensibly diminished in breadth, and that

the closure of it is, *pro tanto*, more likely to be effected than if undertaken in the first instance.

When the palate is entirely deficient, the gap in the soft palate often appears so formidable and unfavorable for operation, that cases have been, not unfrequently, considered beyond relief from the surgeon's hands; and yet there are really very few cases which may not be benefited by operation.

I do not say that the hard palate should be invariably dealt with in the first operation; but the above reasons have induced me to adopt this as the rule; and at present, so far as I can judge, there is every inducement to adhere to it. An additional reason for selecting the operation on the hard palate first, is, that being the most tedious, and by far the most painful one, and as chloroform is not admissible in these cases, it is always something gained in the estimation of the patient, to have the worst the soonest over.

It is essential to union of the flaps, that their edges, when detached from the bones, should touch each other before a single suture is fastened. They should do so by their own weight, and if they slightly overlap it is an advantage, and this without any traction on them by any instrument or suture. This condition is imperative, if union is to take place. One flap may be sufficiently detached, but the opposite one may require a little pressure to make the edges meet; and the pressure of the suture may appear sufficient for this purpose, but it cannot be depended on, as the edges will rarely unite under such circumstances. As surely as the suture exerts the slightest pressure upon the flaps, so surely will it cut itself free; and a line of slough will mark the course of its track.

Before introducing the sutures, a bent probe should be passed round between the flaps and the bone, to ascertain if any point still remains adherent to the bone, and checks the free descent of the flap. This will frequently be the case anteriorly, for in this part it requires much care to release the soft tissue from its attachments; and the ridges of the palate being here most developed and numerous, render it

less yielding and pliant, and more apt to retain any irregularity of surface it has acquired from the frequently irregular condition of the bone. It is of importance to pare the edges before introducing the sutures, for though already raw, they are often irregular, or may have been slightly bruised in their separation from the bone. A clean incised wound offers the best prospect of union, and in this operation we cannot afford to throw away a chance of securing it.

The sutures are passed, as in the operation upon the soft palate, with a common curved needle fixed in a handle. They should not be tightly drawn, for allowance must be made for subsequent tumefaction of the parts. The first should generally be removed on the third or fourth day; the last to be removed should always be the one nearest to any remaining open portion of the cleft.

After the operation food should be given liberally, but in a fluid or pulpy form; and as the patient may wish to avoid taking it, in consequence of the pain sometimes produced by swallowing, the necessity of taking a sufficient quantity must be fully impressed upon him. It is a great mistake to make the patient avoid taking food until union has occurred—solid pieces of food of every kind should be alone avoided. A patient foolishly attempted to eat some pieces of potato a few days after I had operated on the mouth, and accidentally a portion was forced against the freshly united parts, and entirely broke down the union that had taken place.

The tongue will be frequently white and coated after the operation, as if in sympathy with the laceration of the soft parts of the mouth; but it is not usually a condition of importance. I have seen the ill effects of medicines prescribed when this condition has been looked upon as indicating general derangement, and not as one of the effects of the operation.

The conditions of the tongue, in most cases of deficient palate, deserve notice. The surface will be often found in a coated and even dry state in the middle, though it is moist and clean at the edges; in other cases, it will be red

and dry in the centre, though presenting a natural and healthy appearance along its margins. These conditions are readily explained when it is recollected that the middle line of the tongue, in these cases, being opposite the fissure, is exposed to the current of air passing and repassing through the nostrils. The mucous membrane of the upper part of the pharynx will often be found in a similarly dry condition; and sometimes covered with scales of dry mucus, giving to the mouth a disagreeable and unhealthy character.

The health of the patient is a point of the utmost importance at the time of operation. It should be perfect in every sense of the word. For the operation to succeed, union by first intention must take place, otherwise the operation is as abortive as if it had never been attempted. In many operations, the ultimate results are not affected if union does not take place by first intention; but in these cases it is essential to the slightest improvement in the fissure; and unless the patient's health is perfectly good, the operation had better be deferred for a time, if not altogether.

The failure of an operation upon the hard palate, though provoking enough, is no impediment to a second operation on the same part. The soft tissue covering the bones differs in condition a good deal in different individuals; in some, being thick, velvety, and so abundant, that the knife can be freely run between it and the bone; in other instances, being so thin and tightly stretched over the bones, that it is a difficult matter to detach it, and great care is necessary not to cut through it. It is in these latter cases that the failure rather improves the condition of the parts for any secondary attempts to close the fissure, the separation of the flaps having rendered their condition more vascular and substantial.

It has been seen that in fissures extending through the greater portion of the hard palate, the separation of the soft tissues from the bone will generally allow their edges to meet in the median line. But when the fissure does not extend anteriorly beyond the palate bones, then the mere

detachment of the soft parts is not sufficient for that object, and it becomes necessary to make incisions parallel with the edges of the fissure, between the latter and the alveolar margins.

In the latter cases of fissure, the arch of the roof anterior to the cleft is nearly natural, and the sides of the short fissure are inclined inwards, and not perpendicularly. The deficiency of the palate approaches the condition of a foramen; that is, as if a portion of the roof, bone, and soft tissue, had been entirely removed, such as we find to be the case in the diseases which affect these parts.

A simple foramen may exist congenitally in the hard palate, or as the result of disease. The latter is most frequently met with, and when the destruction of the bony palate is not extensive, the conditions may be treated in a manner similar to the fissures which only implicate the posterior portion of the hard palate and the velum. The soft tissues being freely separated round the existing opening, lateral incisions should be made as far from its margins as possible, in order to avoid the chances of sloughing as much as lies in our power. The edges being pared, sutures should be passed into the slips of soft tissue which now lie between the opening and the lateral incisions. An improvement to the support given by these sutures, is obtained by passing a broad suture through the lateral incisions, and then including within its embrace both flaps. This not only serves to support the flaps, but it prevents the healing of the lateral incisions until union has had time to take place between the margins of the opening.

Lateral incisions have been adopted and recommended by most surgeons who have written on this subject, but they have not sufficiently distinguished or pointed out in which class of cases they are essential, or in which they may and should be avoided. As they tend to increase the danger of sloughing, it is most desirable to avoid them, whenever they can possibly be dispensed with.

Plugging the lateral incisions with sponge or lint has also been recommended, but the broad suture answers every

purpose the sponge can effect, and the amount of pressure can be more gradually regulated by it.

The openings resulting from disease may be closed by operation, when the apertures are not extensive; but the treatment is often tedious, and requires much patience on the part of the surgeon.

As deficiencies of the palate are chiefly congenital, and but few the result of disease, it comes to be a question, at what age the operations for closing them should be undertaken. The use of chloroform in this operation is inapplicable, and, consequently, the surgeon requires all the assistance the patient can render him during its performance; and the patient himself must possess a degree of determination for enduring pain, which but few children would be found equal to. For these reasons it will be found, that 17 or 18 is the earliest period at which the operation should be attempted; and also that the delay till this age, having permitted the development of the mouth to become nearly complete, has generally improved the conditions of the fissure for operation. The two specimens exhibited of fissured palate in early life, illustrate how much is to be gained by the prolonged growth of these parts.

But though the operation upon the palate is deferred, the attention of the surgeon may be advantageously turned to the occasional examination of the case; for in the fissures implicating the alveolar ridge, mechanical appliances may be so adapted as to lessen the gap during the period of growth. An instance in which this observation applies, was represented by the model of a case taken from a girl, who was also affected with a most formidable fissure of the upper lip, extending into the nostril. I operated on the lip when she was twelve years old, and subsequently Mr. Vasey prepared for her a plate, to which an India-rubber band was fastened, and so applied round the outer surface of the upper teeth, that the sides of the gap were gently and continuously pressed upon.

In other cases in which the incisor teeth are irregular,

deficient, or supernumerary, the attention of the dentist should always be early directed to their conditions.

The only remaining point for consideration, is the amount of improvement in the voice and articulation likely to be derived from an operation when successful.

Mr. Fergusson remarks that, in most cases in which he had closed the fissure of the soft palate, the distinctness of speech greatly improved, but that occasionally it failed to do so. In a case of deficiency of the hard palate, the imperfection of the voice is generally so extreme, that a sensible improvement will be usually observed even after a partial closure of the gap; but it requires time for any great amelioration to take place. The acquired habit of pronunciation must be altered for a new method of articulating; and great attention is necessary on the part of the learner to repeat distinctly the words spoken by a second person. The best exercise that he can use, is to read aloud to one who will have patience and time to correct his mistakes, and point out his deficiencies.

The difficulties encountered in pronunciation are not in every case entirely dependent on the results of the fissure, even after an operation has been successful. In some cases in which the fissure has extended through the alveolar ridge, it will be found that this imperfection in articulation depends much upon the gap in front, or on the irregularity of the teeth; and that this can only be remedied by the assistance of the dentist,—the point, it may be said, where scientific dentistry comes to the aid of the surgeon. The importance of these features in these cases, and also as regards the personal appearance of the individual, was to some extent illustrated by some models which Mr. Vasey kindly allowed me to make use of.

In presenting these remarks to the consideration of the Society, I have felt considerable difficulty in curtailing my communication within its present limits, without doing a supposed injustice to those who have explored before me the subject herein examined. I allude particularly to the writings of

Roux, Dieffenbach, Warren, Mettauer, Sedillot, and Cloquet. The principles they have recommended for the performance of these operations are said to have been successful, under their own supervision; but if otherwise in the practice of English surgeons, it appeared to me that something was wanting to explain by what means greater success should be ensured by us; and I venture to hope that these remarks will, in some measure, lead towards this desired explanation. I have not entered into the history of the operation, nor have I considered it desirable to repeat all that has been done or proposed in these deficiencies of the hard palate. These points can be referred to by those who feel interested in the subject, but would occupy far too much time in a paper of this character. I have, however, thought it right to place before the profession what may be done in these cases, and the means by which it may be effected, feeling satisfied that few exceptions exist in which the fissures of the hard palate cannot be effectually and permanently relieved by operation.

The chief points to which the surgeon's attention should be directed, to ensure this success, may be thus summed up:

A perfect state of health in the patient at the time of the operation.

Properly adapted instruments for detaching the soft tissues from the bone.

Careful dissection of the soft parts, without bruising or tearing the flaps.

Ample flaps, to enable their edges to meet readily without the traction of the sutures.

Lateral incisions, if necessary, to ensure this condition.

Repeated operations, rather than any one extensive separation of the soft tissues at one proceeding.

Generous diet in a fluid or pulpy form, from the time of the operation until union is firm.

And lastly, perfect rest from all attempts at conversation or speaking, for the first few days after the operation.

A CASE
OF
DESTRUCTION OF THE ENTIRE PALATE,
SUCCESSFULLY RELIEVED BY
MECHANICAL MEANS.

BY
EDWIN SERCOMBE, M.R.C.S.

COMMUNICATED BY
BENJAMIN TRAVERS, F.R.S.

Received Jan. 19th, 1856.—Read Jan. 23d, 1856.

IN one of the earliest volumes of the Transactions of this Society, the President and Council invite "solitary cases, when possessed of peculiar novelty and interest, as deemed by the Society very appropriate objects of communication." Upon this invitation the following case is brought forward, with a hope that though, in some respects, it may not be regarded as a solitary one, it possesses sufficient novelty and interest to commend it to the attention of this Society.

It is intended to omit, on the present occasion, all reference to the early history of this case, and not even to venture an opinion as to what the disease really was, which worked with such destructive energy on the unfortunate

individual who has supplied a text for the following account ; because, interesting as such inquiries might be, it would be beside the point which it is the object of this paper to develop, and which it is of more than ordinary importance to establish, viz., that it is within our reach to restore, by mechanical contrivance, one or more portions of the face, which from disease or injury may have been destroyed.

The following case offered difficulties not likely to be often surpassed, which were increased by an insuperable objection, on the part of the patient and his family, on account of his profession, to the adoption of a beard and moustache, by which the deformity might have been much more easily concealed.

Fourteen years of suffering—always severe, often terrible,—terminated in a rest, which, to the subject of our present remarks, was almost more calamitous than the agony he had endured ; for it was a rest enforced by a disfigurement so great, as to render him altogether unfit to occupy a post for which he was pre-eminently fitted, and to confine him almost within the limits of his immediate family.

Drawing No. 1. represents the face as injured by disease.

Drawing No. 2. represents the face with an artificial upper lip adapted to it.

The external disfigurement is, however, but imperfectly represented by these drawings, for the animation of his eye lent a light, as it were, to bring out the hideousness of a face distorted, contracted, and discoloured by recent disease, but, with this modification, they are perfectly correct.

The work of destruction may be described in few words : the soft parts in the fauces were attacked with the most virulent ulcerations, and sloughed away in masses ; our patient was rapidly reduced from thirteen stone to below ten ; bark and iodine were administered, and he as rapidly recovered weight, gaining twelve pounds in twelve days. The disease, however, was not stayed, the upper front teeth became black, and several were removed ; the gums, instead of healing, ulcerated, and portions of the alvcolar processes

exfoliated. In this way the upper incisors, canines, and bicuspidæ, were lost. The ulceration now crept onwards to the face, the nose was attacked and partially destroyed, the lips completely so; indeed the whole of the face, below a straight line drawn from one meatus auditorius externus over the lower margin of the nasal bones to the other meatus, appears to have been subjected to the same destructive process, for it is intersected in every direction with cicatrices. The intermaxillary and palate bones exfoliated in large pieces; hemorrhage from the palatine arteries, and other smaller branches, lent its terrors to the sufferer, who says, in a note of his case about this time, "very extensive loss of bone of palate and upper jaw, with bleeding from the inside of nose and mouth—excessive distress." The vomer had disappeared. Considerable portions of the inferior maxillary had exfoliated, and all the teeth, save a canine and two incisors, represented in Drawing No. 2, had disappeared.

An external examination of the face displayed, as is shown by the Drawing No. 1., partial destruction of the *alæ nasi*, especially on the right side; complete loss of both lips, as the band stretching across, representing the upper lip, could scarcely be called a lip—it was a firm, unyielding mass of abnormal tissue; thus the cavity of the mouth was at all times exposed through an irregularly oblong opening, represented in the Drawing, on the lower border of which stood the only three teeth found in the lower jaw when I first saw the patient; and evidences of former terrible ulcerations appeared in cicatrices which mapped the face in every direction below the line mentioned above. The contraction of the face during the healing of the ulcerations, had not been less than an inch and a half, measuring from the line where the hair ceased to grow on the forehead to the lower border of the inferior maxilla; and as the shortening of the face was entirely within the lines across the middle of the face above, and the lower border of the inferior jaw below, the face might be said to be very much out of drawing; add to this, that the

greater part of the hole leading into the mouth was to the right of the median line, and we have then a tolerably correct idea of the unsightly appearance of the countenance. In consequence of this contraction, and the rigid condition of the cicatrices, the lower jaw could not be depressed more than the eighth of an inch. The inability to depress it to a greater extent, depended, not upon any injury of the articulation, but upon the unyielding character of the abnormal tissues of the face; for when a free incision was made from the left extremity of the hole towards the left ear, which separated some of the most firm of the bands, considerably increased motion was at once obtained.

On looking into the ori-nasal cavity (for the two were thrown completely into one), and which could be as conveniently done through the right nostril as through the opening below it, it was found to be triangular in shape, with a blunted apex, formed by the lower surface of the cribriform plate of the ethmoid (the central lamella was gone), the base formed by the tongue, and the sides by the superior and middle turbinated bones, lower down by the nasal wall of the antrum of Highmore, on one side by three molar teeth, and on the other by two.

The models and diagrams render any further description of the destruction of parts within and about the mouth unnecessary; we will therefore proceed now to describe the apparatus made. The utmost that was hoped for, when the case was first undertaken, was to render our patient intelligible to his own immediate family and friends; for this purpose it was determined to construct, if possible, some sort of obturator. At this stage of the case, nothing further was contemplated, nor was it until the obturator had been worn some time, that any attempt at making lips was undertaken. The different steps of the task shall be briefly described.

The first step was to remove the three teeth which stood at the entrance of the mouth, for, until this was done, it was impossible to introduce even a single finger; but, although considerable space was thus gained, sufficient in

fact to allow two fingers to be passed together easily into the mouth, yet the want of room to manipulate was so great that I could not refrain from suggesting that an incision be made; and, after consultation with Mr. Travers (under whose care our patient had been for the last three or four years, until the completion of the cicatrizing process and the restoration of his physical powers), it was determined to make one of an inch and a half or two inches long from the left corner of the mouth towards the left ear, and to endeavour to stitch the mucous membrane of the mouth to the skin along the line of incision, so as, if possible, to induce union by the first intention, in which case it was hoped that a permanent opening would be obtained sufficiently large to admit of the introduction of an obturator of the ordinary description. This operation was performed by Mr. Holmes, late house-surgeon of St. George's Hospital; but from the altered state and unyielding consistence of the parts around the opening, it was found impossible to bring the mucous membrane sufficiently close to the skin, union by the first intention did not ensue, and although the incision healed kindly, the contraction was so great that when it was perfectly healed it was found that no increase of room had been gained. It was now obvious that no ordinary mode of treating the case could be employed; for the first step, whether for making artificial teeth or an obturator, is to get a perfect model of the whole jaw, either upper or lower, as the case may be, in softened bee's wax, from which a plaster of Paris model is obtained; but, in the present case, it was absolutely impossible to get in one piece a model of the whole of even one side of what remained of the upper jaw, much less of both; therefore, at the very outset, some new plan had to be devised by which a model could be obtained. The following was the plan adopted: a tray of metal, shaped somewhat like the handle of a spoon, was armed with softened wax, introduced into the mouth and pressed against the sides of the teeth of one side, together with as much of the surface of the bone above them as it would cover, it was then carefully removed, bringing on its surface a correct

impression of the parts with which it had been brought into contact; the same operation was performed on the other side, and plaster of Paris models were obtained from them; but, as the impression thus obtained was not large enough (the extent of it is shown upon the models on the table by a pencil line), it was increased in size by putting a roll of softened wax to the upper border and moulding it with the finger as a sculptor does his clay, until, after two or three attempts, perfect models of the sides, of sufficient size, were obtained. These were again cast in plaster, and to each was fitted a gold plate; but at this point the real difficulties of the case commenced. It was next necessary that the exact distance which these plates would be from each other when in situ should be ascertained; but many and fruitless were the attempts to secure this, until at last a thin plate of Britannia metal, which could be easily moulded by the finger, was carefully fitted against the surface of one of the plates, to which it was attached by a hinge, so that they might be introduced separately into the mouth, and then united by passing a pin through the hinge. This done, the two side plates were first put into their respective places in the mouth, and then the plate of Britannia metal was introduced and secured on the one side by means of the hinge to the one plate in an exact and known position; the other side was then brought against the other side plate and fitted accurately along its irregular surface by simple pressure of the finger, and for further security the line of the Britannia metal plate was scratched by a fine instrument on the surface of that gold side plate to which it was not attached by the hinge. The three plates were now removed separately from the mouth, the two side plates were placed upon their respective models, and the central plate was secured to the one by the hinge on the one side, and on the other side brought to correspond to the line scratched on the surface of the other gold plate, in which position it was secured by softened wax, while plaster of Paris was poured over the whole. In this manner a working model was obtained, upon which the three plates occupied

the exact relative position out of the mouth that they were required to occupy in the mouth. This point once attained, it became an easy task to make a model in plaster which should fill up the intervening space between the two side models, resembling as closely as the case would allow an ordinary palate. On this model the centre plate (a copy of which was on the table) was made; and thus, when the three plates were put together, a complete palatine arch was formed.

The method of securing the three plates firmly together when in the mouth is very simple. The two side plates are introduced separately into the mouth first. The right-hand plate has a groove running along its surface, into which the corresponding side of the centre plate closely fits. The left-hand plate has on its surface one division of a hinge, the other two divisions of which are so secured to the corresponding side of the centre plate that, when it is brought up to its place, which can be accomplished by the tongue, the three divisions of the hinge form one continuous tube, through which is passed a fine gold pin. By this contrivance the three plates are as firmly united when in the mouth as if they were in one piece. The time required to put the apparatus together in the mouth, or to remove it, does not exceed two minutes. With the plates thus arranged the voice at once became considerably improved. Many words could be distinctly pronounced; but there was much yet to be desired. It was, therefore, determined to try the effect of a soft palate. Numberless experiments both in material and shape, which it would be useless to describe here, resulted in one, carved in ivory (a copy of which was laid before the Society), which restored the voice as completely as without lips it was possible to expect. During these experiments one fact in reference to the voice came out most strikingly, viz., that there should be no interruption of continuity between the hard and soft palates; even a pin-hole is sufficient to mar the otherwise good effect, as was found again and again. The importance of supplying lips now for the first time forced itself on my attention. As the

disfigurement was very considerable, it was hoped to cover the whole face below the eyes with a mask, if a material could be found flexible and capable of being sharply moulded and receiving paint; vulcanized India-rubber was found to be such a material. A cast of the whole face was taken, and upon it was built up a face by my friend Mr. Durham, the sculptor, in character with the upper part of the head and face, the junction with which it was intended to hide by the help of spectacles. These casts were copied in type-metal, and forwarded to Manchester, to Messrs. Macintosh and Co.'s manufactory, and the India-rubber mask now on the table was the result. It is defective in several respects in which a second would have been perfect. Its edges are rough and thick, and there is a hole on one side of the nose, all of which imperfections would have been rectified in a second; but it was not obtained, as our patient expressed an insuperable objection to wearing any kind of mask. All he wished to have was lips, so that his voice might be perfected. Lips were therefore made, India-rubber being again used for the purpose. A thin gold band with two hooks was secured to the back of each lip, by which they were to be attached, the upper one to the upper centre plate, and the lower to a plate fitted to the lower jaw, made merely for the purpose of carrying the lip. By this arrangement the lips could be placed and removed at pleasure. With the apparatus thus completed the power of distinct articulation was within a week so far restored that our patient returned to the country to resume duties involving public speaking, from which for years he had been entirely excluded.

The lower lip has been altogether dispensed with, as the plate to which it was attached interfered in some measure with the movements of the soft material covering the chin, which, within the last twelve months, has so gained in fullness and mobility, that there is good reason to hope that in another year a useful natural lip will be formed. The material of the upper lip has been changed from India-rubber to ivory, as it was found that the thin edge of the

rubber decomposed and softened, and the paint scaled off. The ivory has these advantages over the softer material, that the hooks are fixed more firmly, and also that teeth showing below the lip may be riveted to it. The voice is quite as good with the ivory as with the India-rubber lip.

The apparatus thus described has been worn for about nine months, a period long enough to prove it; and it has been found to prevent effectually the passage of fluids from the oral to the nasal cavities during the act of deglutition. The sense of taste has been restored, and appears in no measure defective. The sense of hearing, which was completely destroyed in the right ear and nearly so in the left, is fully restored in the latter; but it remains unimproved in the former. The cause of deafness which the apparatus appears to have removed in so great a degree was, it would seem, the extension through the Eustachian tubes into the middle ear, of a dried and thickened condition of the mucous membrane of the pharynx, brought on by prolonged and direct exposure to the atmosphere at the ordinary temperature.

The power of articulation is so far restored that, in a letter recently received, this patient says, "My voice is as good as I could possibly expect, almost as good as I could wish. I am beginning this new year in the greatest hope and confidence of future comfort and usefulness."

From this case three points appear clearly brought out. 1st. It little matters how long or wide the cleft may be; an obturator can be as easily adapted to the largest as to the smallest opening, therefore the possibility of an operation for cleft palate being followed by sloughing, such as would enlarge the primary opening, ought not to present any grave objection to its being performed; and consequently, in all cases where there is no contra-indication, the chance of a successful issue should be given.

2d. In cases where the fissure is accidental and not congenital, it requires but that the parts lost be artificially

restored, and almost immediately the power of speech is regained.

3d. That portions of the face which may have been destroyed by accidental causes may be restored by mechanical contrivance when beyond the reach of the surgeon. A fact of no small interest at the present time when so many of our countrymen are likely to return home variously mutilated.

CASES
ILLUSTRATIVE OF
THE PATHOLOGY OF THE EAR.

BY
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COMMUNICATED BY
EDWARD STANLEY, F.R.S.

Received Feb. 2d, 1856.—Read Feb. 12th, 1856.

THE series of investigations into the pathology of the ear, laid before the Royal Medical and Chirurgical Society by Mr. Toynbee, have done much to remove the ignorance which previously existed with regard to the diseases of that organ. But the subject is of such extent, and, until lately, has been so little studied, that there is still room for additions to our knowledge. I am therefore induced to bring under the notice of the Society the results of 56 dissections of the ear, made by myself.

The general results of these dissections go to confirm the views advanced by Mr. Toynbee, to whom I am greatly indebted for permission to examine and compare the specimens contained in his museum. A few of them seem to warrant more particular mention.

Of the 56 ears, 12 were healthy. Of the remaining 44 which deviate more or less from the healthy standard, 7 belonged to persons known to be deaf; 9 belonged to persons

known not to be deaf; that is, in whom the power of hearing the voice was observed to be unimpaired, so far as could be judged, to the time of death or loss of consciousness. Of the 28 of which the histories are unknown, 15 present considerable evidences of old-standing disease, and 13 are more slightly affected.

I shall first relate briefly the history of some of the cases; and afterwards present a summary of the morbid appearances detected.

CASE 1.—Deposits, probably tubercular, in either tympanum; caries of the petrous bone on the left side; abscess in the left cerebral hemisphere; erysipelas of the scalp.

J— R—, æt. 19, was admitted into St. Bartholomew's Hospital on the 26th April, 1855. His mother states, that at two years of age he fell upon his head, from a height of two feet. This fall was followed by an illness, during which lumps formed below both ears, and a discharge issued from the left. From that time he has been more or less deaf, and has complained of pain in the left ear, which has been more severe whenever the discharge was not present. On account of his dulness, his father had been much in the habit of boxing his ears. For about a fortnight previously he had suffered from a peculiar shooting pain around the head. On admission into the hospital, the entire scalp was in a state of erysipelatous inflammation, and with it there existed great debility, a small feeble pulse, and profuse diarrhoea. After a few days, suppuration was established beneath the occipito frontalis, and openings having been made at various points, the bones were found to be extensively denuded, being covered by the brawny unadherent scalp. There were no acute cerebral symptoms, but he continued to complain of severe frontal headache. About the 12th of May the discharge from the ear reappeared, having previously been absent for more than a month. The suppuration from the scalp continued, and he was greatly weakened

by occasional attacks of diarrhoea. On the 20th May the headache became more severe, and continued so, but without any fresh symptoms presenting themselves, until he became comatose, about three hours before his death, on the 25th.

Post-mortem examination.—The occipital and the temporal and parietal bones were bare to a great extent, and covered with pus; their surface was dotted over with superficial excavations, giving it a honeycomb appearance. The base of these excavations was rough, and of a pink colour, and many of them were filled with granulations.

The arachnoid was of a dull-yellowish colour, with soft, almost purulent lymph, effused along the track of the principal vessels of the pia mater. The cerebral convolutions on the left side were much flattened. There was a large amount of pus at the base of the brain, and the pia mater, as it dipped in among the convolutions, was covered with thick, puriform fluid, and a similar fluid occupied the anterior cornu of the ventricles, the walls of which were of a bright pink colour, and contrasted with the other parts of the cerebrum, which was, for the most part, unusually pale, as were also the choroid plexuses.

The arachnoid corresponding to that portion of the left hemisphere which rests upon the petrous portion of the temporal bone, bulged before a collection of laudable pus. In all, about two ounces of this fluid occupied, to the destruction of the natural tissue, the middle of this hemisphere, and were contained by a limiting wall of thickened and indurated tissue, highly vascular, vessels being conspicuous running through it in every direction, and an abundance of large exudation cells mixed with filamentous tissue mingling with the constituents of the nervous structure. Thus altered and inflamed, this wall surrounded for a depth of one inch the matter mentioned above; beyond it, for a considerable extent, the cerebrum was quite soft and diffuent.

Both lungs contained numerous groups of small, grey tubercles, the tissue immediately surrounding which was

vascular in excess. The other thoracic and abdominal organs were healthy.

Examination of the petrous bones.—On the left side, the plate of bone forming the roof of the tympanum is of a dirty-brown colour, thin, easily breaking down, and surrounded, in the recent state, by a line of a bright pink colour. The dura mater covering it was separated from the bone, the discoloration of which appeared through it, but it retained its smooth, polished surface. The tympanic cavity, and upper portion of mastoid cells, are full of a soft, greenish-yellow deposit, of the consistence of putty, mingled with some thick puriform fluid, and it communicates with the external surface of the temporal bone by means of a small aperture between the external meatus and the mastoid process. In the situation of this orifice there is a depression in the bone which might contain a bean, and on both the external and internal surfaces of the bone there are irregular elevated patches, arising from the deposition of new bone. The membrana tympani is greatly thickened, and covered with soft, warty growths, so that its natural structure is completely lost. In the upper part, above the head of the malleus, there is a small oval orifice, about two lines in its greatest length. The dermoid layer of the external meatus is much inflamed and thickened.

The right tympanum contains a deposit firmer than that on the left side, and of a dirty-white colour. It occupies the space between the proper cavity of the tympanum and the mastoid cells, and adheres, by many thread-like bands, to the external osseous wall. The fibrous layers of the membrana tympani are thick and opaque; the membrane is extremely concave, its internal surface being nearly in contact with the inner wall of the tympanum.

The general features of this case are such as commonly arise from abscess in the cerebrum, dependent on caries of the petrous bone, but the peculiar direction taken by the ulcerative action, passing through the external plate of the temporal bone, above the mastoid process, is indicative of

disease commencing within the mastoid cells in early life; which the history confirms. Mr. Toynbee has pointed out, that during infancy this plate of bone is exceedingly thin, and forms the external boundary of the horizontal portion of the mastoid cells, which is the only portion then developed. In the left ear, which obviously presents an early stage of the same disease, the morbid deposit occupies precisely that position. The deposition of new bone indicates an effort of nature to repair the injury sustained, an effort rendered abortive by the continuance of the disease in the tympanic cavity, causing caries of the superior wall of the tympanum.

For the history of this case, and the opportunity of examining the temporal bones, I am indebted to Mr. Callender.

An early stage of a disease similar to the foregoing, is presented by the following case.

CASE 2.—Tubercular disease of the brain and lungs; the left tympanum containing semi-purulent mucus, the right tympanum containing a small mass of tubercular deposit.

F—Y—, æt. 3 $\frac{1}{2}$, died March 20th, 1855, after five weeks' illness, of which the prominent symptoms were fever, with obstinate constipation, and diminution of urine, followed by vomiting, pain in the left side of the head, convulsions, and attacks of syncope. On post-mortem examination, the following appearances were found.

The sinuses and membranes of the brain contained a normal amount of blood. On the left side of the middle lobe of the cerebrum, immediately above the fissure of Sylvius, the arachnoid contained numerous small scattered masses of white deposit. These masses covered a space of about three inches vertically by an inch and a half laterally, and appeared nearly to follow the course of the middle cerebral artery. Imbedded in the substance of the left thalamus opticus, immediately beneath this external deposit, was a firm mass of

pale red colour, about the size of a horse-bean, and surrounded by a large plexus of distended blood-vessels.

Throughout the whole cerebrum, but chiefly in the posterior lobes, were scattered small, firm, red masses of deposit, easily turned out from the surrounding cerebral tissue. There were a few in the pons, several in the crura cerebri; none were found in the cerebellum, which was very pale as compared with the cerebrum, but appeared healthy.

Both lungs were thickly studded with tubercles, the kidneys were large and pale. The mesenteric glands were enlarged, and contained tubercular matter.

Examination of the Temporal Bones. Right side.—The membrana tympani was opaque and vascular; the mucous layer much thickened. Around the heads of the malleus and incus there was a soft deposit of pale yellow colour, apparently about three grains in weight. The mucous membrane lining the tympanum and mastoid cells was ecchymosed; the mastoid cells contained a broad false membrane. Red vessels were seen ramifying on the internal surface of the vestibule.

Left side.—Membrana tympani: dermoid layer more vascular than natural; mucous layer thick. The cavity of the tympanum contained puriform mucus; the mastoid cells were filled with a dark-coloured viscid fluid, which appeared to discolour the bone above, and contained also a large red false membrane. Membranous bands also united the incus and stapes together. The mucous membrane lining the tympanum and Eustachian tube was very red and thick. The fluid contained in the tympanum consisted of granular matter mixed with numerous pus cells.

In this case the hearing was very acute during the last illness, and the power of distinguishing the voice appeared to be perfectly preserved to the last. The chief disease within the cranium was found in the immediate neighbourhood of the left petrous bone, and it appears at least possible that irritation, propagated from the inflamed tympanum,

might have acted powerfully as an exciting cause of the fatal tuberculosis of the brain.

CASE 3.—*Eustachian tubes obstructed by thickening of their lining membrane. Cavity of the tympanum on each side containing purulent mucus.*

Samuel H—, æt. 3½, a well-formed child, of dark complexion, was first seen on the evening of 26th May, 1855. He was then in strong convulsions, affecting nearly all the muscles of the body, and resembling an extremely severe form of chorea. He did not appear to be in pain; there was no heat of skin, or excessive quickness of the pulse; he took fluids when desired. He continued convulsed during the whole night, and died exhausted about 9 o'clock on the following morning.

His history, so far as it could be ascertained, was as follows: He was always a very passionate boy, especially during the few last months of his life. He had enjoyed general good health, never complained of headache, but often seemed more unwilling than other children to have his head touched; he also had a habit of putting his fingers in his ears, but never complained of pain in them, nor had there been any discharge from them. Four or five times he has been noticed to throw himself upon the ground, and roll about, apparently in play, yet so strangely as to attract particular attention. Has been deaf, when he has had a cold, for some time. About three days before his death he became much more deaf than ever before, and so continued.

He had appeared in his usual health until the 24th, three days before his death, when he seemed to be languid and ill. On the evening of that day he fell, and struck his head against the door, but not violently, and apparently quite recovered from the effects of the blow. The next day he was feverish, and laid his head upon the pillow. He first became convulsed on the evening of the 26th, and died in fourteen hours afterwards.

Examination twenty-four hours after death.—Appearance of body natural. The sac of the arachnoid contained three or four drachms of clear fluid, and the membranes of the brain generally were much congested. No disease was detected in the substance of the brain, which appeared quite healthy throughout. The spinal cord was not examined. The heart was loosely contracted. A small triangular mass of soft lymph, attached by its base to the apex of the heart, lay in the pericardial sac. There were a few dull white patches on the opposed surfaces of the pericardium, which, however, contained no fluid. Structure of the heart healthy. The lower lobe of the left lung contained scarcely any air; it did not collapse when the chest was opened, nor crepitate beneath the finger. It was of a dark reddish hue when cut into, and broke down on firm pressure. Portions of it sank in water.

Examination of temporal bones.—The mucous membrane of the fauces around the orifices of the Eustachian tubes was very greatly swollen, and infiltrated with muco-purulent fluid, which exuded from it in great quantity when it was pressed. The Eustachian tubes were closed, by approximation of the thickened lining membrane, from the faucial extremity to within half an inch of the tympanic opening. The cavity of the tympanum on each side contained a red-coloured viscid fluid; and the mucous membrane was red, thick, and velvety. The membranæ tympani were fallen in towards the promontory; they were very vascular, and the mucous layer thick.

On the right side, there were many membranous bands uniting the membrana tympani and all the ossicula to the inner wall of the tympanum; these bands existed also in the mastoid cells, and contained many spots of ecchymosis. The cochlea, also (on the right side), was much congested, and contained a red fluid; and red vessels were seen ramifying on the walls of the vestibule and canals.

The starting point of the morbid condition of the ears, in this case, appears to have been the disease of the faucial

mucous membrane extending into the tympanum, and causing obstruction of the Eustachian tubes. Hence arose the accumulation of viscid mucus, and the collapse of the membranæ tympani. This latter condition appears to be an almost invariable result of prolonged obstruction of the Eustachian tubes; as if the want of a renewed supply of air within the tympanum caused the membrane to yield to the pressure of the external atmosphere. How far the morbid condition of the tympana, and on the right side of the cochlea also, might have contributed to induce the convulsive seizure during which the patient died, I have no means of forming an opinion.

The other children of this family, two in number, suffer under deafness, which appears to arise from a similar condition of the throat.

In some cases, it appears that irritation, propagated from an inflamed tympanum, gives rise to cerebral disorder of sufficient intensity to destroy life, without any organic lesion discoverable after death. The following was probably a case of this nature.

CASE 4.—A middle aged man was admitted into St. Bartholomew's Hospital in July, 1854, delirious, and in a moribund condition. The particulars of his history are unknown. On examination after death no disease was found, except that the tympanic cavity and mastoid cells on the left side were full of dark coloured unhealthy pus. The membrana tympani was perforated. The layer of bone forming the roof of the tympanum was dark coloured, and the surrounding cancellous structure infiltrated with pus. The dura mater and brain were healthy. On the right side the lower portion of the mastoid cells and the neighbouring bone were dark coloured, and infiltrated with sanguineous fluid.

CASE 5.—*Accumulation of mucus in the tympanic cavity ; thickening of the mucous membrane.*

Harriet B—, æt. 2 years. Died on 27th January, 1855, from exhaustion, after repeated attacks of bronchitis, accompanied with dilatation and hypertrophy of the heart.

Examination of the temporal bones. Right side.—The dermoid layer of the membrana tympani was more vascular than natural. The mucous membrane of the tympanum was slightly reddened and thick. Firm membranous bands united the incus and stapes to the posterior wall of the tympanum. Red vessels were seen ramifying on the surface of the cochlea and semicircular canals.

Left side.—The membrana tympani was more concave than natural, opaque, of a dull leaden mottled aspect from thickening of its mucous layer. It was covered with red vessels converging from the circumference towards the centre. The tympanum and mastoid cells were filled with mingled purulent fluid and viscid mucus. Membranous bands surrounded the crura of the stapes, and united the incus to the chorda tympani nerve. The mucous membrane was red, velvety, and so thick as greatly to diminish the size of the tympanic cavity, and the diameter of the Eustachian tube. It was also very loosely connected with the bony walls of the tympanum, remaining in its position when the bone was removed. The bone itself, however, was healthy. It would seem probable, that in some cases of caries of the walls of the tympanum, arising from inflammation within its cavity, the cause of the loss of vitality of the bone may be rather a deficient supply of blood, owing to detachment of the inflamed mucous membrane, than a direct extension of the inflammatory action.

In this case no impairment of the hearing had been observed by the parents ; but it appeared, on inquiry, that the child had for some time been in the habit of putting

her hands to her ears, and the mother had frequently picked them with a pin, which seemed to give her pleasure.

CASE 6.—*Deafness on the left side. Membranous bands in either tympanum ; ossicula on the left side drawn together, and less moveable than natural.*

H. W—, a tall, apparently robust man, died suddenly at the age of 52. The muscular structure of the heart was in a state of degeneration.

History.—He suffered from ear-ache in early life, and was noticed to be slightly deaf at about 26 years of age. For some years past he had been unable to hear without great difficulty with the left ear, and when spoken to would habitually turn the right ear. He was able, however, to carry on ordinary conversation with perfect facility, the hearing on the right side being very good. He had always been subject to headache, and three years ago had an illness pronounced to be congestion of the brain.

His mother became deaf in both ears at 7 years of age from scarlatina, and her deafness increased as she advanced in life. All his brothers and sisters, of whom there were four, suffered in childhood from pain in the ears. His sister, aged 40, is very deaf, the symptoms indicating an affection of the auditory nerve. His two brothers are deaf in a less degree.

Examination of the petrous bones.—The left ear, in which the hearing was impaired, presented the following appearances: The membrana tympani was slightly opaque, thicker and more concave than natural, and rigid. The mucous membrane of the tympanum was red and thick. The ossicula were approximated to each other, and firmly bound by membranous bands, uniting the long crus of the incus to the membrana tympani and to the handle of the malleus, and the stapes to the other ossicles and tympanic walls. The stapes was firmly fixed in the fenestra ovalis,

being immoveable without considerable pressure. The vestibule and semicircular canals were healthy. The cochlea more vascular than natural. The membrana fenestræ rotundæ was opaque.

Right ear.—The meatus externus contained a small accumulation of soft cerumen. The membrana tympani and mucous membrane of tympanum healthy. The ossicula were surrounded by membranous bands, connecting the membrana tympani and malleus to the long process of the incus, and the stapes and incus to the posterior wall of the tympanum. The ossicles retained their natural mobility. The internal ear was healthy, except a slight vascularity of the cochlea and adjacent part of the vestibule.

Adhesions of the ossicula to each other and to the walls of the tympanum are forms of disease of such frequent occurrence, that great interest attaches to any facts that can throw light upon the influence they exert upon the faculty of hearing. In the foregoing case they exist to about an equal extent on each side; but with this important difference, that in the ear of which the hearing was impaired, the chain of bones is drawn together and rendered immoveable. In the other ear the ossicula retain their proper positions and natural mobility. This case, therefore, affords evidence that membranous bands within the tympanum do not interfere with audition unless they impede the motions of the ossicula, or cause the stapes to press upon the fluid of the vestibule.

In the following case it is probable that the contraction of membranous bands in the tympanum diminishing the mobility of the ossicula, and especially of the stapes, was a cause of deafness.

CASE 7.—A female aged 8 years. Died from dropsy after scarlatina. Was observed to be hard of hearing during her last illness.

Examination of temporal bones.— On each side the meatus



externus contained desquamating epidermis. The membrana tympani is healthy in structure; but more concave than natural. All the ossicula are united to one another and to the walls of the tympanum by their transparent bands. The heads of the malleus and incus covered with red vessels. Stapes less moveable than natural. Internal ear healthy.

Mucus accumulated within the cavities of the ear will sometimes dry up after death into membrane-like films, which present a very similar appearance to adhesions formed during life. This was observed in the following two cases :

CASE 8.—*E. F—, male, æt. 17. Died, after eight months' illness, of phthisis complicated with mania. His hearing appeared good to the time of death.*

Examination of temporal bones.—The membrana tympani on the right side was very concave, its inner surface being nearly in contact with the promontory. It was covered with thickened epidermis, and the circular fibrous layer was also opaque and thickened to a slight extent. The left membrana tympani was more concave than natural; but its structure was healthy. The cavity of the tympanum on each side contained numerous fine bands uniting the ossicula to each other and to the posterior osseous wall. Broad bands existed also in the mastoid cells. The mucous membrane of the tympanum and the internal ear on each side were healthy. Much thick mucus was collected about the upper part of the pharynx, and the faucial extremity of the Eustachian tubes was filled with transparent viscid mucus. This mucus, having been suffered to remain in the Eustachian tube of the left side, had dried into fine transparent bands, stretching across the tube (which had been laid open), and precisely resembling the bands which existed within the tympanum.

CASE 9.—*Ann D—, et. 2. Died from whooping cough complicated with diarrhœa. Hearing not impaired.*

Examination of the temporal bones. On the right side the mucous membrane of the tympanum was much congested, of a pinkish hue, and covered with a thin layer of mucus. The cochlea also was congested.

On the left side the epidermis covering the membrana tympani was thick; numerous red vessels ramified in the dermoid layer. The mucous membrane of the tympanum was of a bright red colour, the ossicula were covered with red vessels, and the long process of the incus appeared of a bright pink hue through the membrana tympani. The base of the stapes in each ear formed a strong contrast, by its red colour, to the other parts of the wall of the vestibule. Stretching across the mastoid cells on the left side was a layer of semifluid tenacious substance, of a dirty white colour, which was converted, in the course of a few days, into a firm transparent pseudo-membrane containing red streaks, and closely resembling the membranous bands so frequently found in the same situation.

The tympanum being a cavity containing air which is frequently renewed, it is conceivable that the drying up of mucus during life might give rise to one form of the membranous adhesions which constitute so frequent a pathological appearance.

Examined beneath the microscope the dried mucus presented an agglomeration of granular matter and shrivelled cells of varying thickness and tenacity. A portion of some firm adhesions formed during life presented a distinctly fibrous structure.

Membranous adhesions in the tympanum are not only extremely common, being present in 29 out of 56 dissections, but they are met with also in very early life. One of the specimens exhibited was the right temporal bone of a child, aged 4 months, who died with symptoms of secondary sup-

puration. It exhibited a broad transparent band uniting the posterior crus of the stapes to the canal of the portio dura nerve.

The following is a short statement of the morbid conditions found in the 44 diseased ears.

I. Eight ears belonging to persons known to be deaf.

MEATUS EXTERNUS.

Healthy	3
Containing desquamating epidermis	3
Dermoid layer inflamed, and thickened from chronic inflammation	2

MEMBRANA TYMPANI.

Concave, and less moveable than natural	2
Concave, opaque, rigid, mucous layer thickened	1
Collapsed, vascular, mucous layer thickened	2
Fibrous layers thickened	1
Perforated and thickened	1
Perforated, and covered with fungoid granulations	1

CAVITY OF THE TYMPANUM.

Mucous membrane healthy, containing membranous bands	2
Mucous membrane red and thick, containing membranous bands	1
Mucous membrane red and thick, containing membranous bands and mucus	2
Mucous membrane red and thick, containing pus	1
Mucous membrane red and ecchymosed, containing scrofulous matter	2

MASTOID CELLS.

Healthy	3
Mucous membrane congested	3
Containing bands and ecchymosis	1
Containing pus	1

OSICULA.

Healthy	2
Membrane covering them red	1
United by membranous bands	4
Imbedded in scrofulous matter and pus	2
Stapes less mobile than natural	2

EUSTACHIAN TUBE.

Closed by thickened mucous membrane	2
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COCHLEA.

Congested	1
Congested, and containing red fluid	1

VESTIBULE AND SEMICIRCULAR CANALS.

Congested	1
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MEMBRANA FENESTRÆ ROTUNDÆ.

Opaque	1
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II. Nine ears belonging to persons known not to be deaf.**MEATUS EXTERNUS.**

Healthy	5
Containing desquamating epidermis	1
Containing cerumen and epidermis	1

MEMBRANA TYMPANI.

Healthy	2
Epidermoid layer thick	2
Dermoid layer vascular	2
Circular fibrous layer thickened	1
Vascular: mucous layer thick	2

CAVITY OF THE TYMPANUM.

Mucous membrane healthy; containing membranous bands	4
Mucous membrane congested	2
Mucous membrane thick and red	1
Mucous membrane thick and red; containing mucus, and membranous bands	1
Mucous membrane red and ecchymosed; containing scrofulous matter	1

MASTOID CELLS.

Healthy	3
Congested	1
Congested, and containing membranous bands	2
Healthy, and containing membranous bands	3

EUSTACHIAN TUBE.

Small, mucous membrane thick	2
Containing viscid mucus	2

VESTIBULE.

Congested	2
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COCHLEA.

Congested	2
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III. Twenty-seven ears; the histories unknown.**MEATUS EXTERNUS.**

Healthy	16
Containing cerumen	2
Containing cerumen, and thickened epidermis	3
Containing cerumen; dermoid layer red	3
Containing exostosis	1
Lower bony wall deficient	1
Affected with erysipelas	1

MEMBRANA TYMPANI.

Healthy	7
Concave	1
Concave and rigid	1
Epidermoid layer thick	3
Dermoid layer vascular	4
Fibrous layers opaque	2
Opaque, relaxed, pressed in by cerumen	2
Relaxed, concave, dull	1
Drawn in, and adherent to incus	1
Opaque and mottled, from thickened mucous layer	1
Containing calcareous deposit	1
Containing calcareous deposit, and perforated	2
Perforated, the edges of the orifice attached to the incus and walls of tympanum	1

CAVITY OF THE TYMPANUM.

Healthy	4
Mucous membrane congested	1
Mucous membrane congested and thickened	4
Containing adhesions	6
Mucous membrane thickened, containing adhesions	4
Mucous membrane thick and red, containing adhesions	2
Containing red lymph and adhesions	1
Containing a sanguineous fluid and adhesions	3
Containing pus and adhesions, mucous membrane thick and red	1

MASTOID CELLS.

Healthy	12
Congested	2
Containing adhesions	4
Containing mucus	2
Containing pus	1
Sanguineous fluid effused in the cancellous tissue around tympanum (typhus fever)	2
Bone around the mastoid process infiltrated with fluid and of dark colour	3

OSSICULA.

Healthy	5
United by adhesions	18
Red vessels ramifying over them	5
Ditto and ecchymosis	1
Less mobile than natural	1
Deposit of red lymph on base of stapes	1
Malleus partially detached from membrana tympani	1
Stapes united by membranous ankylosis to margin of fenestra ovalis	3
Stapes dislocated into vestibule at posterior border	1

EUSTACHIAN TUBE.

Very large	1
Contracted by approximation of bony walls	1
Thin spiculæ of bone growing from its floor	1
Mucous membrane thick and red, containing mucus	1
Closed by thickened mucous membrane	1

COCHLEA.

Congested	1
Congested, the contained fluid of a red colour	2
Lamina spiralis broken down: the scalæ towards their termination containing a granular fibrinous exudation, covering the fenestra rotunda	1

SEMICIRCULAR CANALS.

Red vessels ramifying on their surface	2
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MEMBRANE OF FENESTRA ROTUNDA.

Opaque	2
Opaque and small	1
Opaque and thick	1
Covered by false membrane on its tympanal aspect	1
Covered by fibrinous deposit on its cochlear aspect	1

AUDITORY NERVE.

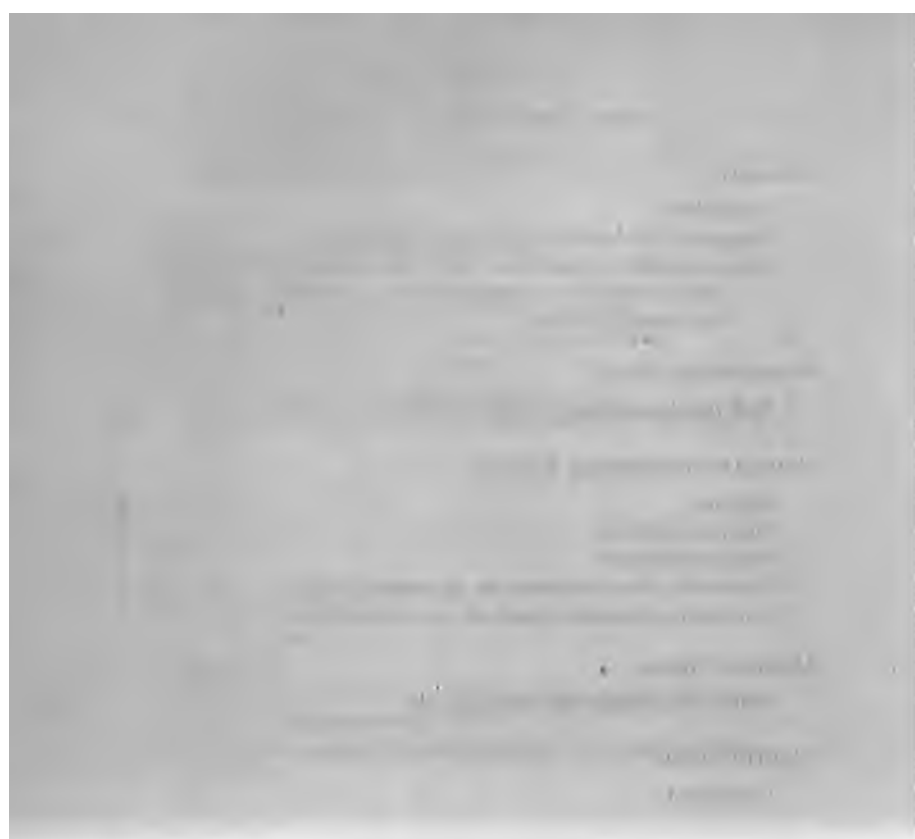
Red at its entrance into the vestibule	1
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CAROTID CANAL.

Contracted	2
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I would venture to call the attention of the Society to the very large proportion of cases in which disease of a decided character has been found. The ears have been taken indiscriminately from subjects of all ages, yet only 23 per cent. are healthy. Nor is it unimportant to observe that in two instances in which the tympanic cavity in children has been found in an advanced stage of inflammatory disease, there existed no symptoms during life by which special attention would have been directed to the ear.

And, in conclusion, I would express my concurrence in the opinion pronounced by Mr. Toynbee, that "investigations into the nature and treatment of diseases of the ear will be fruitful in valuable results if they are steadily pursued in an earnest and patient spirit."



ON

MYELOID AND MYELO-CYSTIC
TUMOURS OF BONE;

THEIR STRUCTURE, PATHOLOGY, AND MODE
OF DIAGNOSIS.

BY

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THERE are probably few subjects in surgical pathology that present so great an amount of interest as the different forms of tumours, and more especially those which attack the osseous tissue. The varied form of structure which they present, the difficulty attending their diagnosis, and the great importance of the part they affect, inasmuch as their existence in it, in some cases, involves the removal of part or of the whole of the affected limb, necessarily gives them an amount of interest far greater than attaches to those which arise in other tissues. It is from these circumstances that I have been induced to lay before this Society the results of some observations that I have made upon some forms of tumours of bone, which, as far as I am aware, have been but very slightly noticed by surgical writers. It is well known that

there are some forms of tumours of bone that present, in every respect, so marked a resemblance to hæmatoid and medullary cancer, as far as the naked eye examination goes, that it is almost impossible to distinguish them from it. Sir Benjamin Brodie, in his last edition on the diseases of the joints, was the first, I believe, who called attention to this form of disease. In a statement he makes of an examination of a limb amputated for disease in the femur, he observes:¹ "On examining the limb afterwards, the femur was found to terminate abruptly, about five inches above the knee-joint. In place of the condyles, and lower part of the shaft of the bone, there was a large tumour, of irregular form, the structure of which bore a nearer resemblance to fungus hæmatodes than to any other morbid growth." And further on he says: "It may be observed, that the structure of the tumour is described not as being that of fungus hæmatodes, but as bearing a nearer resemblance to it, than to that of any other morbid growth. It would appear, therefore, that I must, in the first instance, have entertained some doubt as to the exact nature of the disease. To whatever extent these doubts existed, subsequent observations have satisfied me that they were not without foundation, and have, indeed, led me to the conclusion, that the disease was not fungus hæmatodes, but a peculiar change in the structure of the femur, of local origin, and not, in the proper acceptance of the term, malignant."

Now, although Sir Benjamin Brodie doubts the similarity of these tumours to hæmatoid cancer, no further observations were made by him of their structure. The account, however, that I shall give of these cases will, I think, clearly prove that they are not of a malignant nature, but that they represent in their structure a kind of overgrowth of some of the normal constituents of bone, originating, probably, from some local cause; thus bearing out the opinion first enunciated by Sir Benjamin Brodie, "that the

¹ 'Pathological and Surgical Observations on the Diseases of the Joints,' by Sir B. C. Brodie, Bart., fifth edition, p. 275.

disease was not fungus hæmatodes, but a peculiar change in the structure of the femur, of local origin, and not, in the proper acceptation of the term, malignant."

Some cases of tumours of bone, to all appearance of a malignant character, have occurred, in which the entire bone has been removed by operation. In these cases, the disease not having recurred in other parts of the body, the line of practice has been adopted of removal of the bone in the joint above the diseased part, in nearly all cases, as a preventive measure against the return of the disease, and the non-occurrence of the disease for some years after seemed to warrant the practice adopted.

In one of these cases that I have examined, of disease of the upper end of the humerus, in which the bone was removed at the shoulder-joint, under the supposition that the disease was of a malignant nature, I have found its structure precisely similar to those above mentioned, consequently the conclusions drawn from this case, as to the probable success of a like operation, in cases of malignant disease, is negatived.

Again, some forms of tumours of bone, to all appearance of a malignant nature, have existed, in which the patient has died previous to any operation, and in which, on a post-mortem examination being made, no similar disease has been detected in any other part of the body. Such cases have been thought favorable instances of a malignant disease confined to the bone alone, and consequently favorable cases for operation. In a tumour of such a kind that I have examined, supposed to be of malignant character, the structure was precisely similar to those mentioned by Sir Benjamin Brodie, and the disease was not of a malignant nature. The conclusions consequently drawn from this case, as to the probable success of an operation in a case of malignant disease of bone, is also negatived.

I have made these observations, in order to show the extreme interest that must attach to a class of tumours bearing so close a resemblance to malignant disease, that the eye alone fails to detect any difference between them—

the importance of being able to diagnose their structure and properties,—the doubt that must now necessarily arise in our minds as to the accuracy of some reported cases of malignant disease of bone,—and the conclusions drawn from them, as to the question of amputation, and the ultimate result of the disease.

It is probable that these tumours were first received under the head of sarcomatous or osteo-sarcomatous tumours.

At a later period, M. Lébert described a class of tumours, including these and other forms, under the name of “fibro-plastic,” and showed the similarity which they bear to malignant disease.¹ More recently, Mr. Paget described the fibro-plastic tumours of Lébert under the name of myeloid, from the circumstance of their characteristic constituent consisting of large nucleated corpuscles, exactly similar to the same forms that have been described by Kölliker and Robin, as a normal constituent of the marrow of bones, especially in the fœtus, and at an early period of life. He showed, also, the close affinity they bear to cartilaginous and osseous tumours, from their structure being occasionally mixed with cartilage and normally developed, well-formed, cancellous bone.

In the account which I shall now give of these tumours, I hope to be able to show :

1st. That the essential element consists, in all cases, of forms precisely similar to what is found in the marrow and other elements of bone in the fœtus, and at an early period of life; hence the name “myeloid” tumours. In some instances, however, their structure is so intermixed with cysts, that I would propose the term “myelocystic” tumours to be given to them in such cases.

2dly. That these tumours are, for the most part, limited in their development and growth to the osseous tissue, or its investing membranes, the periosteum and dura mater.

3dly. That they may probably occur in any bone.

¹ ‘Physiologie Pathologique,’ par H. Lébert, tome ii, p. 120, 1845.

4thly. That they occur in all the cases at present recorded at an early period of life,¹ and that their growth is generally much less rapid than that of malignant disease.

5thly. That these tumours are not malignant, and when entirely removed never return.

6thly. That they present a near relation with fibrous and fibro-cystic tumours, and cartilaginous and osseous.

Before detailing any account of the cases of this disease that I have examined, I am anxious to state that none of them were recent specimens, but had been preserved for some years in alcohol; I have no reason, however, to believe that their structure has been otherwise altered than by the loss of colour which they had possessed in their recent state.²

CASE 1 (Plate 1).—The following notes of the history of this case are taken from the case-book of Sir Benjamin Brodie, who has permitted me to make use of them:

“Miss H— consulted me, in the spring of 1825, on account of an enlargement of the head of the tibia, attended with little pain, and producing slight impediment to the motions of the knee. The tumour gradually increased, still producing but little pain, the knee retaining its mobility up to the day of amputation, which was in September, 1827. Several glands in the neck of this patient were enlarged, and she suffered from cough. She died ten days after the operation; and on examining the body, there were no marks of internal disease, except a small cyst of the ovarium, containing about an ounce of dark coloured fluid. On examination of the limb, the knee-joint was perfectly free from disease, the synovial membrane and cartilages assuming their natural appearance.”

¹ I have recently had an opportunity of examining a specimen of this disease from a female of middle age.

² Since the above was written I have had the opportunity of examining several recent specimens of the disease; the general appearances, as well as the minute structure, were in every respect similar to those which had been immersed in alcohol.

In a more accurate examination that I have lately made of the part, I found that the upper portion of the shaft terminated abruptly about four inches below the head, and in place of the head and upper part of the bone, was a large tumour of a somewhat rounded shape, contained in a cyst formed of periosteum and a layer of cellular membrane; the bone in which the tumour was developed being absorbed in every part. The periosteum which covered the head of the tibia was in every part much thinned, but in no part had it been entirely absorbed. The tumour consisted of a large mass of solid matter, of a greyish-white colour, with a flocculent surface, the chief part of which lay free in the cavity in which it was contained. It was of a firmer consistence in some parts than in others, very vascular, and presented patches of a buff-yellow colour disseminated through it, which indicated the existence of advanced fatty degeneration. The shaft of the bone was somewhat abruptly broken where it joined the substance of the tumour; and the medullary canal, for about half an inch in its extent, was filled with a deposit similar to that of which the tumour was composed, but the bony tissue was not infiltrated with it.

The structure of the tumour differed somewhat in different parts. At its upper part, immediately beneath the cartilage of the tibia, where its structure was firmer and more dense, it was composed entirely of a mass of variously shaped fibres. Some of them were of an acicular form, and contained nonuclei; others were of a spindle shape, containing chiefly rod-like or oval nuclei, with granular contents, or occasionally a few refractive oil globules. All the softer and more flocculent portions of the tumour consisted of large cells and fibres. The fibres were similar to those above mentioned as composing the firmer portions of the tumour. The cells varied much in their size; some were about the size of the normal medulla cells, others five or even ten times that size; their form was rarely circular, more frequently oval or oblong; the cell wall in some cases was very distinct, in others less so; occasionally they presented branched or caudate fibres proceeding from

one point of their circumference, or they were encased in foldings of these spindle-shaped fibres, arranged concentrically around their outer surface. Their contents also varied; some containing a few circular or oval-shaped nuclei, with central nucleoli, the rest of the cell being full of fine granules; in others the contents of the cell presented so dark an appearance, that the individual elements occupying the interior could not be detected. Others, but these were few in number, contained many oil globules, or presented a delicate transparency, as if the contents of the cell had become liquified or discharged.

Such were the various elements composing this tumour, and it will be seen that they differ in every respect from those which are peculiar to the varied forms of cancer.

The entire absence of cystic cavities, and the fact of its being composed throughout of solid matter, form interesting points in this case. In the remaining cases to which I shall allude, cysts were combined with the solid matter in varying proportions, in the second case being few in number, whilst in the third and fourth, they formed the chief part of the whole mass.

CASE 2 (Plate 1).—The following notes of the history of this case are transcribed, by the permission of Sir Benjamin Brodie, from the last edition of his work on the 'Diseases of the Joints,' p. 272.

"In June, 1826, I was consulted respecting a young lady, about 18 years of age, who laboured under a considerable enlargement of one shoulder. The head of the humerus was expanded into a broad and somewhat elastic tumour. There was some, but not considerable, pain in the joint. The mobility of the spine was necessarily impaired from the increased size of the humerus, but did not seem to be affected otherwise. In a consultation between Sir A. Cooper and myself, it was agreed that the limb should be removed at the

shoulder joint, which operation I afterwards performed in the presence of Sir A. Cooper and Mr. Aston Key.

"The disease was wholly confined to the head of the humerus, which was converted into a medullary or fungous tumour of considerable size, with very little remains of earthy material in it.

"The scapula, the cartilage lining the glenoid cavity, the capsular ligament, and synovial membrane of the joint, were in a healthy state. The cartilage covering the head of the humerus remained entire, adhering to the surface of the morbid growth. The wound healed readily, and I know that there had been no return of the disease two years afterwards."

In a minute examination that I have recently made of the diseased part, I found that it consisted of the development of a large tumour in the head and upper part of the shaft of the bone. The tumour was of an oblong rounded form, about the size of a small cocoa nut, and of a moderately uniform surface externally, it presented a grayish white colour, and consisted of two distinct portions, solid matter and cysts. The whole circumference of the upper end of the bone was involved in the disease, from the articular surface of the head, which still remained, to the commencement of the upper third of the shaft, which was abruptly broken immediately below the tumour. The tumour was invested with a thick fibrous capsule, which was continuous with the periosteum. The shaft of the bone, where abruptly broken immediately below the tumour, had, bulging into its medullary canal, for about half an inch of its extent, a quantity of growth similar to that which composed the mass of the tumour, and with which it was directly continuous: immediately below the point where the fracture had occurred a quantity of new bone had been deposited on the external surface of the shaft, but separated from the old bone by a protrusion of the same material in the interspace between them.

The solid matter of the tumour was of a yellowish white colour, firm, solid, and somewhat brittle in texture. Its structure was precisely similar to that of the preceding case.

There is one fact, however, of very considerable importance to be learned from the naked-eye examination of these two tumours, viz., the circumstance of the remaining part of the shaft of the bone in both cases not being infiltrated with the disease.

It is well known that in the great majority of cases of malignant disease of bone, and more especially of that form which arises in the cancellous tissue, that the shaft of the bone, for a considerable extent above the part where the disease is most developed, is extensively infiltrated with a similar deposit. In these cases, however, the disease does not infiltrate the substance of the bone, and it is evident that the growth which filled the upper part of the medullary canal, for about half an inch in both cases, is to be explained by the protrusion of the morbid structure into it, which resulted from the diminished amount of resistance offered to its progress at that part.

In the third case, the history of which I shall now relate, the disease also commenced in the articular extremity of one of the long bones. In this instance, however, the tumour had in its growth approached so near to the surface of the joint, as to cause inflammation to take place in it, and numerous soft bands of adhesion might be seen to exist between the cartilaginous surfaces, which prevented the extension of the growth into its cavity.

CASE 3 (Plate II).—For the notes of this case I am also indebted to the kindness of Sir Benjamin Brodie, who has permitted me to transcribe them from his work on the ‘Diseases of the Joints,’ p. 274.

“Mr. O—, æt. 25, in January, 1828, first experienced a sensation of weakness in the right knee, with a slight pain, after walking even a short distance. These symptoms continued; and, in the course of two or three months, he observed a small tumour over the external condyle. He remained in this state, the tumour not increasing in size, through the spring and the greater part of the summer.

In the middle of the following August he one day went through a great deal of fatigue in grouse-shooting, after which the tumour began to increase in size. On the 1st of September, in walking over a field, his foot slipped into a hollow in the ground. This caused great pain in the knee, and he was under the necessity of riding home. After this accident the tumour progressively increased. On the 25th of January, 1829, he came to London, and placed himself under the care of Mr. Griffiths, of Pimlico, and myself. At this time there was a very considerable enlargement of the whole of the upper part of the knee-joint, so that it was four inches more in circumference than the corresponding part of the opposite limb. The tumour was soft and elastic, occupying the situations of both condyles of the femur, but being more especially prominent in that of the outer condyle. The head of the tibia and the patella did not seem to be implicated in the disease, and the joint retained its natural degree of mobility.

"For some time after I was consulted the tumour remained nearly stationary: then it began to increase; and, as no remedy seemed to have any dominion over the disease, a consultation was held with Sir Astley Cooper, in which it was determined that the limb should be amputated. The operation was accordingly performed on the 6th of July, 1829.

"I heard of this patient being alive and well five years after the operation, having had no recurrence of the disease.

"On examining the limb the femur was found to terminate abruptly about five inches above the knee-joint. In place of the condyles and lower part of the shaft of that bone, there was a large tumour of an irregular form, the structure of which bore a nearer resemblance to fungus hæmatodes than to any other morbid growth. The cartilage which had covered the surface of the condyles of the femur was seen expanded over the lower part of the tumour, being everywhere thinner than natural, but nowhere in a state of ulceration. In some parts it had contracted adhesions to the cartilages covering the head of the tibia and side of the

patella. In other parts the tumour was covered by some thin remains of the periosteum and a layer of thickened cellular membrane, which forms a kind of cyst in which the growth is contained."

In a careful examination that I have recently made of the diseased part, I found the medullary canal, where abruptly broken off at its connection with the tumour, filled up by a nearly solid mass of newly formed cancellous bone, so that the growth did not project into the canal, nor was there any infiltration of the cancellous texture at that part.

The tumour consisted of solid matter and cysts, the minute structure of which was precisely similar to that last described.

The description of this case accords in nearly every respect with the two already recorded. In all, the structure of the morbid growth presented the same characteristic elements. In all, the bone terminated abruptly at the point where it was connected to the tumour. In all, the structure of the shaft was not infiltrated with the disease, and in the clinical history of this case it has been shown that, although amputation of the limb was performed through the bone only a short distance above the diseased point, no return of the disease was known to have occurred for several years afterwards.

In the next case which I shall relate, it will be seen that the morbid growth, which also commenced in the articular end of one of the long bones, had made its way into the neighbouring joint, which it distended throughout, absorbing by its pressure the articular surfaces of all the bones forming its cavity, and protruding into the substance of the bones in many parts.

CASE 4 (Plate II).—For the particulars of this case I am also indebted to the kindness of Sir Benjamin Brodie.

"— B—, æt. about 36,¹ was carrying a heavy burden, when his right foot became entangled in a hole in the ground, and he fell. He immediately experienced a severe pain in the knee, and it was after this accident that the enlargement of the joint was first observed. The tumour continued to increase, with a severe shooting pain, until it was twenty-six inches in circumference. Six years from the time of the first appearance of the disease I amputated the thigh, in St. George's Hospital. I have since heard of the patient, who was alive and well, having had no recurrence of the disease several years (ten) afterwards."

In a recent examination that I have made, I found that the tumour, which was nine inches in length, and twenty-six inches in circumference, commenced at the lower part of the shaft of the femur, just where it joined the condyles; externally, the surface was deeply uneven, and covered by a strong fibrous investment, the remains of the periosteum, and, in parts, by a thin shell of bone, the remains of the condyles of the femur, which were spread over the surface of the tumour. The articular surface of the condyles had been entirely absorbed, and the morbid growth had made its way into the cavity of the knee-joint, which it distended throughout, pushing the patella and its ligament forward, absorbing the cartilaginous surface of that bone, and making its way into the cancellous tissue. The cartilage covering the head of the tibia had the growth adhering to it in some parts, whilst in others the cartilage had been absorbed, and the morbid structure had made its way into the cancellous tissue of this bone, but only to a very slight extent. At the back part of the tumour a deep groove in its substance received the popliteal vessels and nerves. Just at the point where the shaft of the bone joined the morbid growth, a thick outgrowth of bone had been developed, which was intimately adherent to the shaft, and projected over the upper part of the tumour. Nearly the whole anterior half of the tumour presented an opaque-white colour, was very firm in texture, and of a tough, leathery consistence, composed entirely of

¹ The exact age of this patient I have not been able to ascertain.

wavy fibres, like ordinary white fibrous tissue, with imbedded nuclei of an elongated oval form. These fibres were arranged into bands and masses, of different forms and sizes, which enclosed cystic cavities, varying in their size from a millet seed to a walnut. In every respect this portion of the morbid growth corresponded, in its general appearance and structure, with the ordinary forms of fibro-cystic tumours. The whole of the posterior half of the tumour was of a buff colour, it was softer in texture, and tore up more readily, than the other portions of the growth. In general appearance, and in its minute structure, this portion of the tumour was analogous to the tumours in the cases previously recorded.

The cysts had a wall formed of a fibro-granular lamina, without an epithelial lining, and contained either serum,—or the remains of effused blood,—or fibrin, as clearly ascertainable by the peculiar fibrillation of the contained fibrin, and the existence of large, granular lymph corpuscles,—or degenerate fibrin, in the form of granules or granular masses of a circular form and of an oleo-albuminous composition.

This tumour presents many points of considerable interest; the combination of the myeloid and fibro-cystic structures in one tumour; its extremely slow growth; its extension into the cavity of the joint; the non-infiltration of the morbid structure in the shaft of the bone above the diseased part, and its non-recurrence ten years after removal, are facts of great importance, inasmuch as most of them present a marked contrast to malignant tumours.

CASE 5.—For an examination that I have recently made of this preparation, I am greatly indebted to the kindness of Mr. Stanley and Mr. Paget, who allowed me the freest access to the rich collection of tumours of bone contained in the Museum of St. Bartholomew's Hospital.

The details of the case are described by Mr. Lawrence, in a paper entitled, "Observations on Tumours, with Cases," in vol. xvii of 'The Medico-Chirurgical Transactions,' and was apparently regarded by him as a disease of a mixed

kind, but of a decidedly malignant character. The tumour was connected with the upper end of the tibia; its growth was of eighteen months' duration previous to amputation: it appeared after a fall, and was occasionally very painful. It occurred in a female, 30 years of age.

In a recent examination that I made of this tumour, I found that the soft substance which presented so close a resemblance to medullary cancer was composed of the same elementary structures that have been described and figured in the preceding cases.

CASE 6.—For the notes of this case I am indebted to the kindness of Mr. G. Borlase Childs.

Harriet H—, æt. 23, was admitted into the Royal Free Hospital, under the care of Mr. Childs, on 4th December, 1855, with an enlargement of the lower part of the right thigh. To the eye, the disease much resembled a chronic swelling of the joint itself, the enlargement being about equal in all parts, and evenly rounded. On looking more carefully, however, it might be noticed that its boundaries, above and below, were too abrupt for joint-disease. The head of the tibia just below the overhanging mass, and the leg itself, were free from any disease. The finger confirmed this impression, the patella being movable, and without any degree of swelling beneath its ligament. The lower fifth of the femur, on the contrary, was felt to be involved in a hard, bone-like enlargement, twenty inches in circumference, which had greatly expanded its condyles, but which decreased above rather gradually. The overlying skin was somewhat thickened, but not in the least inflamed. The joint was moveable, though not so much so as natural, and no pain was complained of in the examination. The inguinal glands were not enlarged, and there were no symptoms of malignant disease of the internal organs. The history she gave was, that in March, 1853, she began to feel occasional weakness in the knee, which would at times "give way," in walking, and cause great pain. Some months after the aching began, a little enlargement on the outer side was noticed, but no

great increase in size took place until March, 1854, when the tumour rapidly increased in size for about two months, and then became almost stationary. Whilst increasing in size a great deal of pain was present, and the least jar to the leg used to cause a wrenching pain. When quiet, there was no material aching; but for all this, at times she was kept awake by it. In June, 1854, she was sent to the Margate Sea Bathing Infirmary; during her residence there she improved in health, but the size of the limb increased, according to her statement, an inch and a half in circumference. In March, 1855, she became, for the second time, an inmate of the London Hospital, the limb retaining the same size and general appearance. The summer of 1855 she again spent at Margate, but without benefit. It will be observed that no increase in the size of the tumour occurred during the eight months prior to the amputation: its circumference, in December, being exactly what it had been in March.

On December 10th amputation of the thigh was performed. A section made vertically through the bones constituting the knee-joint showed the tibia and patella quite healthy, the joint also free from effusion, and, excepting a few tough bands of old adhesion here and there, in a normal condition. There was no absorption of the cartilage, but that covering the condyles of the femur was in some parts very thin.


From a careful examination that I have lately made of this tumour, it would appear that the morbid structure had taken its origin in the lower extremity of the condyles of the femur, and that in its growth it expanded the compact surface of the entire circumference of the bone into a shell, which, with the periosteum, formed a complete cyst, in which the morbid growth was contained. There is one point, however, in which this tumour differed from any of the preceding ones, namely, in the manner in which the shaft of the bone above the diseased part was connected with it. In all the cases previously recorded, the shaft of the bone was abruptly broken off immediately beyond the disease; in this

specimen this abrupt termination of the bone did not exist, but the lower end of the shaft, for about the extent of three inches, was somewhat compressed from before backwards, and formed part of the anterior solid wall of the tumour, leading one to the conclusion that the tumour had in the first place originated in the articular end of the bone, but in its growth had projected upwards and backwards behind the lower end of the shaft, which it compressed forwards so as to occupy the situation already mentioned. The section of the tumour consisted of cysts and solid matter.

The cysts varied in their size from a marble to a small orange; their interior was in all cases perfectly smooth, their wall consisting of a nuclear fibrillated membrane, in some cases of considerable thickness, without any epithelial lining.

The chief mass of the solid matter consisted of a soft substance, pulpy in consistence, of a light brownish-red or salmon colour, interposed between the cysts, and occurring more or less in irregular and variously shaped isolated masses. This substance was precisely similar in structure to that in the other cases already recorded.

Besides the reddish pulpy structure above mentioned, the tumour in part consisted of a mass of soft, cream-white or yellow pultaceous substance, pulpy and granular in consistence, and evidently the *débris* of the degenerating element of the tumour. Certain portions of the tumour presented a number of isolated patches of fibrin of a perfectly white colour, and from their structure evidently of varying dates of growth. In two separate parts of the tumour these masses of fibrin were deposited in the interior of the cysts. They were soft in consistence, and in their texture and general appearance resembled in every respect fibrin recently deposited on serous surfaces as a result of inflammation. There were also large masses of the same substance, but they were evidently of a much older date, being firm and solid in texture, and presenting the ordinary structure of pure white fibrous tissue. These masses were of a rounded or irregular form, varying in size from a hazel nut to a walnut.



Bone occurred, also, in the substance of the tumour; 1st, as an isolated mass of small size, and 2dly, in the form of ossification of the fibrous boundary of one of the cyst walls.

The result of this examination would lead me to regard this tumour as a specimen of myelo-cystic, among which large masses of fibrous tissue in various degrees of development were deposited, the myeloid structure being in some parts normal, in others presenting indications of fatty degeneration.

The above are the only cases in which I have been able myself to examine these tumours. Mr. Paget has, however, kindly placed at my disposal the notes of another case (Case 7) of the same disease, a detailed account of which is given in his work on 'Tumours,' and which forms the only additional one that I have been enabled to collect, in which the morbid growth has arisen in the interior of a bone, and has at the same time presented a similarity with, or has been mistaken for, malignant disease; and in which also I have been able to obtain a clear history of the origin, the progress, and the termination of the disease.

I think I have shown, in the preceding cases, that the morbid growth may consist entirely of solid matter, or that the solid matter may be mixed with cysts, or with a fibro-cystic growth.

I shall now relate the histories of two cases in which these tumours were combined, in one case with cartilage, and in another with cartilage and bone: and I detail them, not merely to show the varieties their structure may present, but also to exhibit the affinities which they bear in this diversity of structure to all the primordial elements of bone.

CASE 8.—For the account of this case I am also indebted to Mr. Paget, who has most liberally placed the notes of it at my disposal, and from them the following account is abridged:

The tumour, which involved the lower end of the fibula and external malleolus, was sent to Mr. Stanley by Mr. Langston Parker, and was removed from a young man, 19 years old, whose leg was amputated for the disease. The swelling had been observed twelve months, and had increased slowly without giving much pain. No cause could be assigned for it. During its existence the tumour pulsated synchronously with the arteries, but only in that half of it which was next the tibia. It measured about three inches by one and a half. The walls of the cyst were partly osseous, partly fibrous. The substance of the tumour was firm, for the most part cream white, like firm medullary cancer, but blotched and shaded with blood colour, and having imbedded in it small isolated nodules of cartilage of irregular form.

The structure of the non-cartilaginous portion was precisely similar to what has been observed in the previous cases.

The patient from whom this tumour was removed recovered from the operation, and was known to be well in September, 1854, four years from the period of its removal.

CASE 9.—In the 'Medical Times' for the year 1852, page 214, an interesting case is recorded (with accompanying illustrations) by the late Mr. Bransby Cooper, of a diseased growth, the principal characteristic features of which were the coexistence of the myeloid cells with cartilage and bone.

There can be hardly any doubt, from the description of this tumour, that it was a myelo-cystic growth, containing masses of cartilage, and bone in progress of development.

It will be observed that the structure of the morbid growth which I have described was precisely similar in the first three cases. In the first case, it consisted entirely of solid matter; in the second and third, of solid matter intermixed with cysts. In the fourth and sixth cases the growth was

mixed up with pure fibrous and fibro-cystic structure; in the fifth, with bone; in the eighth, with cartilage, and in the ninth, with cartilage and bone.

I propose now, in the first place, to consider—Of what nature are these tumours? Are they malignant, or are they analogous in their structure to some of the normal textures of the body?

The tumours were composed of solid matter, in some cases mixed with cysts. The solid matter consisted of fibres and cells.

The fibrous element differed somewhat in its structure in different parts of the same tumour, and in different specimens, according to its date of development, growth, and decay. In the 2d case, where the morbid structure had been most recently formed, the fibres were variously shaped; some were of an acicular form and contained no nuclei, others were of a spindle shape, containing chiefly rod-shaped or oval nuclei with granular contents. In this stage of their development it would appear that in some cases the fibres degenerate in their structure, and present an advanced condition of fatty degeneration, as in the 1st case, where large masses of buff or light yellow coloured deposit showed the fibrous elements of this portion disseminated with numerous oil globules, or presenting a finely dotted granular degeneration and breaking up of the individual fibres. On the other hand the fibrous element in some cases advances in its development and becomes converted into simple fibrous tissue, presenting similar appearances to those observed in this tissue in normal textures. Such an advanced condition in the development of this element of the tumour was observed in a portion of the tumour in the 3d case, and also in nearly the whole of the anterior half in the 4th.

The chief and most important constituent in these tumours consists of cells of large size and very peculiar form. They vary in size from the $\frac{1}{350}$ th to the $\frac{1}{1600}$ th part of an inch in diameter, those of large size being by far the most frequent.

They vary also in form. Occasionally they are circular; more frequently oval, or oblong, or flask-shaped; more rarely the outline is lobed like a terminal vesicle of a gland; and in some cases they present well-marked caudate prolongations of large size. Their cell wall in some cases is clear and well defined, in others the contents are arranged in a cell-like mass without any investing membrane. Their contents also vary. Most frequently they consist of numerous circular nuclei, which vary in number from four or five to ten, twenty, or even more. They are occasionally transparent, spherical in form, with central nucleoli, and these are blended with a finely granular substance. Frequently the contents of the cells are dark, from the coagulation of the contained material. The proportion of the cells to the fibrous element of the growth also varies; more frequently the cellular forms predominate, in some parts they may preserve a nearly equal proportion, or the fibrous element may be more marked. Occasionally, in those parts of the growth where it is evident that degeneration of the structure is proceeding, these cells may be observed to contain numerous oil globules, or the cells dwindle in size, their walls collapse, and their contents disappear. These, together with a few free nuclei, similar to those contained in the larger cells, and from which all the stages of the development of the fibrous element may be traced, make up the constituent solid parts of this morbid growth.

The cystic portion of these tumours appears to form a large part of them. In one of the present cases, however, cysts were wanting, the whole tumour being composed of solid matter. These cysts vary considerably in their size, often being as small as a millet seed, or as large as a small apple; they vary in number from three or four, to forty or fifty. Their walls consist of a delicate fibro-granular lamina, not epitheliated, in the texture of which a plexus of blood-vessels is distributed. Their contents vary; occasionally they are filled with a serous fluid, or blood-tinged serum, or coagulated fibrin, the composition of which is ascertained by

the peculiar fibrillation of the fibres and their co-admixture with granular lymph corpuscles.

Such are the essential elements of which the morbid growth constituting the greater part of these tumours is composed.

The next and most important point for consideration is as to whether these elementary forms are such as are met with in cases of malignant disease, and whether the history of the morbid structure,—its mode of growth, its rate of progress, and ultimate result,—is such as to justify us in classing it amongst this formidable class of diseases.

It would appear that the naked-eye examination, (however well versed the observer may be) is quite incapable, even with the greatest care, of detecting the true composition of these morbid structures. This is admitted by all. Sir Benjamin Brodie remarks, "Its structure bore a nearer resemblance to fungus hæmatodes than to any other morbid growth." Mr. Paget also, in a case lately recorded by him, observes, "None who examined this disease with the naked eye alone felt any doubt that it was an example of medullary cancer, with cysts abundantly formed in it." It is only by minute investigation, such as is afforded by the microscope, that its structure, as I have described, can be detected. It is hardly necessary here to observe that these forms do not bear the least resemblance to the varied cellular and fibre forms that are found in malignant disease. But I consider this test to be one of the slightest weight in determining the malignancy of these tumours, when compared with the more important ones which have already been mentioned in the history of these growths; and perhaps there is no test of more practical or diagnostic interest than is obtained by the comparison of the duration of the disease in these cases, with that in undoubted malignant disease. Of the nine cases recorded in this paper, in eight of which the duration of the disease previous to operative interference has been noted, one had existed a year, two a year and a half, one two years and a half, two three years, one six years, one

twelve years. The average duration of the disease, then, in these eight cases, previous to any operation, was nearly four years; a period considerably greater than even the duration of life, with or without operative interference, in malignant disease.

Again, also, in this disease the duration of life after operation is seen to be far greater than after any form of malignant disease in which the diseased part has been removed. Of the nine cases here recorded, in six of which the duration of life after the operation was known, three were known to be alive two years afterwards, one four years afterwards, one five years, and one ten years. Of the three remaining cases, one died from the effects of the operation; and in the other two, the cases were recorded soon after the operation, and the result, as to the duration of life, or recurrence of the disease, is unknown. In all the cases, also, that have been recorded, there has been complete absence of all infiltration of the morbid growth in the shaft of the bone beyond the diseased part, a circumstance of most common and almost constant occurrence in malignant disease. In all these cases the lymphatic glands have been unaffected, there has been complete absence of the malignant cachexia, and the removal of the limb, although in many of these cases through the bone in which the disease had originated, has been followed by no return of the disease after many years.

These are facts of the very highest practical importance, and when coupled with the minute examination of the morbid growth,—presenting, as it does, a marked contrast to the varied forms found in cancer,—I think we are justified in considering that no one of these tumours, either in its minute structure, or in its history, presents the least resemblance to malignant disease.

If this disease, then, be not malignant, which normal texture of the body does it most resemble?

Kölliker and Robin have lately shown that the medullary cavities of bone in the foetus, however they arise, are filled

with a soft, reddish substance, the foetal medulla. This substance at first consists of nothing but a small quantity of fluid, and many rounded cells, with one or two nuclei, and faintly granular contents. In process of time, these cells are developed in the usual way, into connective tissue, blood-vessels, fat-cells, and nerves. In the adult it has been shown that these cells only normally exist in certain bones, namely, the articular extremities of the cylindrical bones, the vertebrae, the true cranial bones, in the sternum, and in the ribs, and apparently in variable number in the bones of the face. Kölliker, also, in speaking of the elementary processes in the development of the layers of bone formed from the periosteum, observes, that the bone-cavities of the periosteal layers, besides medulla-cells and vessels, contain round, elongated, or dentate, flattened, faintly granulated cellular corpuscles, with from three to twelve or more vesicular nuclei and nucleoli, which are probably referable to the multiplication of the medulla-cells. These last-mentioned cellular forms are in every respect identical with those forming one of the constituent parts of the morbid growth in the tumours under consideration.

In the cancellous tissue of the shaft of a femur, of a foetus of seven months, I found, besides the small medulla-cells, a few large circular-shaped cells containing from eight to ten circular nuclei with nucleoli. I examined the same part in a child of six months, as well as the epiphysal end of the femur. In the shaft I saw a few large and variously shaped cells, in every respect identical with those found in the structure of the morbid growths. One of these was of an oblong or club-shaped form, of very large size, and contained from eight to ten circular nuclei with nucleoli; others, also of large size, presented several caudate prolongations proceeding from various parts of their circumference; but neither of these forms abounded. Similar cells were found in very considerable numbers in the lower epiphysal end of the femur of a young man, 19 years of age, whose limb had been amputated for inflammation and abscess

of the bone which led into the knee-joint. The cancelli of the epiphyses were filled with them ; they were of unusually large size, of very irregular form, and crowded with nuclei. A fourth time I found them in the cancellous tissue of the epiphysis of the femur of a young woman, 18 years of age, whose limb had been amputated for disease of the tibia. In other cases, at the same period of life, where the cancelli were full of fat, I have failed to detect them. At a late period of life I have never seen them.

These large many-nucleated cells were perfectly transparent, so that their contents were clearly discernible, and in this respect they presented a marked contrast to those which form a constituent part of these tumours. On the addition of alcohol to them, they immediately lost their transparency, became opaque, their contents darkly granular, and to such an extent as to obscure the nuclei, which, previous to the addition of this reagent, were so well marked. This experiment was repeated several times and with the same result, which convinced me that the dark granular appearance of the many-nucleated cells composing the tumours was derived from their immersion in alcohol, which coagulated the albuminous contents of the cells.

These facts would seem to show that these cells are essential constituents of the medulla of the bone, during the most active periods of growth, and that when this is completed they degenerate into the fat with which the cancelli are filled.

It would appear, then, from the result of these investigations that these tumours, so far at least as one of the elementary forms composing them is concerned, consist of an abnormal amount of some of the normal constituents of the medulla,—the medulla-cells ; hence the name “myeloid” tumours which has been given to them ; and from their being occasionally mixed with cysts, I would propose the name of “myelo-cystic” tumours ; the fibrous element which they present is most probably derived from the organization of lymph effused as a result of chronic inflammatory action, or from some

abnormality in the development and growth of the fibrous element of bone.

Now, if it be admitted that these tumours are analogous in their structure to some of the normal constituents of the medulla, it will be necessary to show—

1st. That they are confined to the osseous tissue or its investing membranes.

2dly. That they occur at the period of life in which the above-described forms exist in normal bone.

From M. Lébert's article on Fibro Plastic Tumours (in his '*Pathologie Anatomique*'), under which title tumours in every respect identical to the "myeloid" are included, it would appear at first sight that they occurred in various situations. If, however, such cases only are included as are figured and described by forms similar to those detailed by me in this paper, it will be found to embrace only those growths which are connected with the osseous tissue or one of its investing membranes, the periosteum, and dura mater.

Mr. Paget, following M. Lébert, has also stated that these tumours may be found in many situations; but all the cases he describes are connected with the osseous tissue, excepting two, in both of which he doubts the true "myeloid character" and suspects a malignant nature, which their history and termination would appear to prove.

In the nine cases which have been recorded in this paper, the disease has occurred between the ages of 18 and 36. In the five cases also recorded by Mr. Paget the variation in age has been between 15 and 24 years.

It would appear, then, that these tumours occur at an early period of life and within the period in which the cellular forms above mentioned form a constituent part of the normal medulla.

It is a remarkable circumstance, also, that in every one of these nine cases the tumours have taken their origin in the epiphysal ends of the long bones.


It has thus been shown that these tumours, in their minute structure, bear a very close analogy with the normal constituents of the marrow of the bones; a fact, too, which is corroborated by the circumstance that they are confined to the osseous texture, that they occur at the period of life when such constituents of the medulla exist to the greatest amount, and that they are developed in those parts of the osseous system in which such structures exist in a most distinct and well-marked form. The fact that these growths are occasionally mixed with some of the other elements of bone in its rudimentary state, as fibrous tissue and cartilage, and even of those of bone itself, serves to make this analogy more complete.

I shall now make some few observations on the pathology and mode of diagnosis of these tumours.

The number of cases at present collected is probably too small to determine if either sex is more liable than the other to this form of disease. Of the nine cases recorded in this paper, six occurred in females, three in males. In the four recorded by Mr. Paget, two occurred in males and two in females.

It is difficult to assign a local cause in all cases, so as to account for the origin of this disease; but of the nine cases here collected, there were two very clear cases of history of severe injury, followed immediately by swelling, which did not subside, but constantly increased, and where there was evidently no interval between the assigned cause and the appearance of the disease. In four other cases no cause whatever could be assigned, and in the two remaining ones, the history affords no clue to enable us to determine whether any local cause existed or not.

When it is known that the mean age of the occurrence of medullary cancer is at an early period of life, and that the bones, in the great proportion of cases, are the organs which are particularly the seat of the disease, it becomes a



very important point to discriminate, if possible, between "myeloid" or "myelo-cystic" disease and medullary cancer, on account of the different operative measures that are required in the two cases.

In the nine cases recorded in this paper, there are eight in which the duration of the disease has been noted. In one case it was a year, in two cases a year and a half, in one two years and a half, in two cases three years, in a fourth case six, and in one case twelve years. Now, if these periods are compared with the duration of life in medullary cancer, a very striking difference is observed. From a table of fifty cases of medullary cancer collected by Mr. Paget, in which the disease pursued its course without operative interference, the average duration of life was rather more than two years, the latest period being four years. Again, in forty-six cases, also collected by the same author, in which medullary cancers were removed by excision or amputation, the average duration of life was something more than twenty-eight months.

The long duration of the disease, as compared with medullary cancer, affords, then, a very important diagnostic mark, more especially when it is considered how closely osteoid and malignant tumours resemble it in their general form.

Other minor diagnostic marks of importance may be also noticed—the absence of the malignant cachexia, so marked a feature of medullary cancer, even while the local disease is of a trivial nature; the absence of lymphatic glandular enlargement; and the absence of disease in the internal organs.

The chief results of these investigations, I think, clearly prove that there are a class of tumours which have their origin either upon, or in the interior of, the bones, and which bear so close a resemblance to malignant disease, that the eye fails to detect their true character. Minute examination of them, however, shows them to consist,—partly, of structures in every respect identical with some of the normal

constituents of the medulla—partly, of a fibrous element mixed up with cysts of varying number and size—and, occasionally, of fibro-cystic structure, or some of the primordial elements of bone itself.

It would thus appear, that non-malignant tumours of bone may consist of structures which are composed entirely of a tissue analogous to one of the normal elements of bone, or of some, or all, of these tissues intermingled. Thus, the fibrous element of bone may be represented by the development of a fibrous tumour, growing either upon or in the interior of the bone, and consisting either entirely of solid matter, or mixed up with cysts. Again, the cartilaginous, osseous, or vascular elements may be represented by the development and growth of one of these structures from the surface, or from the interior of a bone. I think, also, the cases and observations contained in this paper, prove that the *medulla* element of bone may be represented by the development and growth of a tumour, which, in its structure, may be analogous to the cellular structure found in the normal medulla, and consist either entirely of solid matter, or of solid matter intermixed with cysts, and be mixed up, in some cases, with some or all of the primordial elements of bone, as fibrous tissue, cartilage, or bone, in various proportions.

It also appears that these tumours are confined in their growth (as far as we are at present aware) to the osseous tissue, or its investing membranes; that they occur at an early period of life, and bear some relation as to their period of growth to the time of life when the medullary cells are most active, and exist in large proportion. It has also been seen that, although the local cause which has given rise to their development has been traced, in some cases, to a distinct injury, still in many no cause whatever can be assigned for their origin. That their growth is slow, extending in many cases over many years, accompanied by pain, varied in intensity in different cases, attaining occasionally a very considerable size, but in no instance presenting any tendency

to ulcerate or protrude externally ; and presenting, lastly, a marked contrast to malignant disease, in their microscopic structure, in the non-infiltration of the bone from which the tumours take their origin, in their slow growth, in the absence of lymphatic glandular enlargement, and of the extreme wasting and pain and sallow aspect, so marked in most cases of malignant disease. Lastly, the non-recurrence of the disease in any case, during many years after the removal of the limb, is a still more conclusive proof of their innocent character.

I cannot conclude this communication without thanking Sir Benjamin Brodie, Bart., Mr. Stanley, and Mr. Paget, all of whom, in the most kind manner, placed at my disposal their notes of such cases as I required, and allowed me the freest access to their preparations.

My thanks are also especially due to my friend Mr. Vandyke Carter, to whom I am indebted for the illustrations appended to this paper.



RATE OF
HOURLY PULSATION AND RESPIRATION
IN PHTHISIS,
AND
ITS RELATIONS TO SLEEP, FOOD, SUNLIGHT, &c.
WITH FOUR DIAGRAMS AND TABLES.

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THE following is part of an extended investigation, now in progress, at the Hospital for Consumption and Diseases of the Chest, Brompton.

The subjects were three men, who had already aided in another inquiry, and three women, and were placed in two wards apart from other patients. The rate of pulsation and respiration was ascertained at the commencement of each of 144 consecutive hours, or six days and nights, in the lying posture only. No interference with their habits was permitted, except that of lying down at five minutes before and a few minutes after each hour. Their rest, at night, was

commonly undisturbed. The meal hours were $8\frac{1}{2}$ a.m., 5 p.m., and $8\frac{1}{2}$ p.m., for both sexes; and $12\frac{1}{2}$ p.m., for the women, and nearly 1 p.m., for the men. The hour of rising was usually $8\frac{1}{2}$ a.m., and of retiring $8\frac{1}{2}$ p.m. No complication occurred, except a slight attack of diarrhœa in No. 80, and of hæmoptysis in No. 73.

The respirations were counted in full minutes, and the pulsations in half minutes, and during the night the former were counted by the ear, so as to avoid disturbing the patients by the touch. The touch, in counting the pulsations, occasionally awoke the patients, and, on such occasions, we determined the different rates of the functions during sleep and waking.

Nos. 73, 80, and 88, were males; and Nos. 33, 34, and 55, were females. The following are the physical signs in each case:

No. 73 was æt. 22, 4 ft. $9\frac{3}{8}$ in. high, of sanguine temperament, and a compositor. He had been ill ten months, and was much emaciated. His vital capacity was 90 C. I. Right lung: dull universally, prolonged expiration, less vesicular murmur, cavity. Left lung: dull universally, moist râles, prolonged expiration, very little vesicular murmur, bronchial respiration.

No. 80, æt. 20, 5 ft. $5\frac{1}{4}$ in. high, spare, active, an in-door servant, ill at intervals for years, aspect nearly healthy. Right lung: dull (moderately), patches of crepitant and other moist râles. Left lung: dull, flattening, very little mobility, less respiration, less vesicular murmur, two cavities.

No. 88, æt. 45, 5 ft. $6\frac{3}{4}$ in. high, sensitive, spare, a ship-joiner, ill eighteen months, emaciated, vital capacity 76 to 100, C. I. Right lung: dull down to fifth rib, cavity to fourth rib, moist râles, no vesicular murmur, little bronchial respiration. Left lung: dull over the clavicle, prolonged expiration, less vesicular murmur.

No. 33, æt. 40, married, ill twelve months, emaciated. Right lung: dull to second rib, harsh respiration, prolonged expiration, very short inspiration, no vesicular murmur, crepitation to fourth rib, cavity. Left lung: dull

to second rib, prolonged expiration below, no vesicular murmur above.

No. 34, æt. 22, married, dressmaker, ill twelve months, but little emaciated, very excitable. Right lung: slight crepitation and prolonged expiration down to third rib. Left lung: dull to third rib, cavity at apex, crepitation to fourth rib, prolonged expiration, less vesicular murmur.

No. 35, æt. 41, widow, needlewoman, ill twelve months, emaciated. Right lung: dulness, prolonged expiration at the apex, less vesicular murmur. Left lung: dulness (great), less mobility, depression and cavity in second space, no vesicular murmur above, and lessened below.

Thus the ages were 20, 22, 22, 40, 41, and 45, and all the cases were in the stage of softening and formation of a cavity, but they took food and exercise well and slept well.

Four diagrams illustrate this paper. Nos. 1 and 2 exhibit the rate of both functions at each of 144 consecutive hours, or six days and nights; whilst No. 3 shows the results for each hour, on an average of the whole period, in each case. In No. 4 the average hourly variations of respiration in each case, and in all the cases of each sex combined, and of the temperature of the wards, are recorded.

The first part treats of the pulsation, considered first absolutely, and then in relation to various disturbing causes, and the second part, of the rate of respiration, considered in a similar manner. The quantity of air inspired, and the chemical changes effected at various periods of the day, are under examination.

I am greatly indebted to Vertue Edwards, Esq., our resident surgeon, for having taken the observations from 5 to 12 p.m., inclusive.

PART I.
PULSATION.

A. THE HOURLY CHANGES IN PULSATION, ABSOLUTELY:

These are shown in each case in Diagrams Nos. 1, 2, and 3.

On the average the pulse was the lowest from 1 to 5 a.m., and the highest from 10 a.m. to 9 p.m. There was an elevation of 26 pulsations per minute from 5 to 10 a.m., and a corresponding but lesser subsidence, in an equal period, from 9 p.m. to 1 a.m. From 1 to 5 a.m. the pulse was under 68; at 10 a.m. 91; at 3 p.m. 94; and at 6 and 7 p.m. $92\frac{1}{2}$; whilst at midnight it was 76, when it was 8 beats above the minimum of the twenty-four hours. The lowest point during the day-light was mid-day, and then it was 10 beats above the pulsation at midnight. Between the hours of lowest pulsation at night the range was only 3 pulsations, and the highest points of the day, viz., 10 a.m., 3 p.m., 6 p.m., and 9 p.m., did not differ from each other more than 5 pulsations. After 5 a.m. the pulse rose, whilst the patient was yet asleep, more when he awoke, at about 7 a.m., still more on rising, and most of all directly after breakfast. During the day, it attained its highest point in the hour following the meal, and then subsided to a much lower point before the next meal. This was due to the meals, and therefore the hours of highest elevation, as also the height of the elevation, and the depth of the succeeding depressions, depended upon the hour and frequency of meals. At about 8 p.m. or 9 p.m., according to the influence of the supper, the pulse fell whilst the patient was yet up, but more so on going to bed, and still more so when asleep, so

that the first minimum hour was about the fourth after retiring to rest.

The cases varied greatly in the number of pulsations, but agreed as to the periodic variations now given, as shown in the following table.

TABLE NO. I.

Averages for each case for the whole week, and total averages in the two sexes.

Hour.	Cases.			Men's Average.	Women's Average.	Cases.		
	73.	80.	88.			33.	34.	35.
12	91.6	74.0	73.3	79.6	73.4	65.3	77.1	78.0
1 a.m.	88.8	63.3	70.1	74.0	68.9	57.8	76.6	72.3
2 "	86.5	68.1	69.0	74.4	68.5	57.9	76.1	71.5
3 "	80.7	64.1	66.5	70.4	67.2	56.0	76.6	69.2
4 "	82.3	70.7	64.6	72.5	67.8	55.3	75.6	72.6
5 "	80.0	63.6	65.6	69.7	66.7	57.0	72.1	71.1
6 "	82.1	74.1	65.6	73.9	69.5	60.1	74.2	74.3
7 "	84.6	76.1	74.0	78.2	72.6	64.0	77.5	76.5
8 "	83.1	72.6	75.6	77.1	78.8	66.5	82.0	87.8
9 "	95.6	80.0	93.0	89.5	83.0	74.1	81.8	93.3
10 "	104.0	84.3	94.3	94.2	88.5	75.8	94.0	95.8
11 "	103.1	81.8	86.1	90.3	87.6	79.0	90.0	93.8
12 "	97.5	81.6	84.0	87.7	85.7	77.8	87.3	92.0
1 p.m.	98.1	82.6	85.0	88.5	89.4	80.3	91.5	96.5
2 "	98.1	87.5	89.6	91.7	92.7	84.6	94.6	99.1
3 "	103.8	93.1	90.6	95.8	91.6	85.5	91.5	98.0
4 "	100.6	89.8	85.8	92.0	90.1	80.7	92.4	97.3
5 "	97.0	84.6	87.0	89.5	90.6	85.0	92.0	95.0
6 "	102.1	85.0	89.3	92.1	92.5	83.8	94.6	99.3
7 "	103.3	90.8	87.5	93.8	91.1	82.3	94.6	96.6
8 "	101.1	87.5	84.8	91.1	90.2	81.6	95.5	93.5
9 "	95.6	84.3	87.3	89.0	88.5	78.6	89.3	97.6
10 "	93.0	81.0	83.8	85.9	85.4	79.3	83.3	93.6
11 "	89.0	77.6	77.6	81.4	81.2	75.3	79.3	89.0

Range of Pulsation at various periods of the day.

The term "range" expresses the difference between the highest and lowest observations within given periods; and the inquiry into it is as important in reference to the functions of respiration and pulsation in man as it is in meteorological inquiries.

The range of the whole 24 hours varied from 22 to 45 pulsations, both extremes being found in the same case. The average was 34 pulsations in both sexes.

The whole day was divided into four periods, viz., "night," from 1 to 5 a.m.; "day," from 9 a.m. to 9 p.m.; "morning," from 5 a.m. to 10 a.m.; and "evening," from 9 p.m. to 1 a.m. In the first the pulsation was at its minimum, and in the second at its maximum, whilst it rose in the morning and fell in the evening. Each period will be considered separately.

1st. "Night." The average range varied from 1 to 32 pulsations in the different cases, and from 7 to 32 in the same case. It was much greater in males than in females, being 12 in the former, and only $8\frac{1}{2}$ in the latter. Thus the pulse varies in the quietude of the depth of the night through an average of 10 pulsations.

2d. "Day." The total average variation was 17 pulsations, and it was less in women than in men, viz., 15 in the former, and $18\frac{1}{2}$ in the latter. The extremes were 10 and 35 in different cases; but, upon the whole, there was much more uniformity in the "day" than in the "night" pulsation.

3d. "Morning." This was very great, and on the average was not less than 27 pulsations, or 25 in the women and $28\frac{1}{2}$ in the men; thus equaling the united ranges of the "day" and "night." The extremes were 12 and 44, but the increase of pulsation was more uniform than those numbers indicate.

4th. "Evening." This is the only period in which the range of pulsation was greater in women than in men,

viz., 20 in the former, and $17\frac{1}{3}$ in the latter. The average was 19 pulsations, which was less than that of the morning, but more than those of the "day" and "night." The extremes were 8 and 32 pulsations.

Thus the four periods occupy the following order in reference to the range of pulsation, viz., "night" 10, "day" 17, "evening" 19, and "morning" 27 pulsations. The ranges of "morning" and "evening" are nearly twice as great as those of the "day" and "night." The extremes of pulsations were the greatest in the "night." Thus the causes which influence pulsation must be more active in the "morning" and "evening" than in the developed "day" and "night." In women these influences produce a more rapid evening subsidence of the pulse, and a lower pulsation through the night than in men.

The Hours and Amount of Minimum and Maximum Pulsation.

Minimum in the night.—The total average was 65 pulsations, and was less in women than in men. The extremes in all the cases were 52 and 81, but there was great constancy in each case separately, the variations being to the extent of 5 pulsations only. There is no relation between the minimum pulsation and extent of disease. The hour of lowest pulsation was most frequently 5 a.m., and then 6 and 4 a.m.

Minimum in the day.—This, on the average, was 84, and was nearly the same in both sexes. It was 20 pulsations above the night minimum. The extremes were considerable, viz., 65 and 100, and in each case it was greater than in the night minimum. The lowest day and the lowest night minimum were not always met with in the same case. The proportionate increase of the day over the night minimum was greatest in women, but this increase was not uniform. Thus taking the night minimum as the basis, the day increase varied as follows: Men over $\frac{1}{4}$, about $\frac{1}{4}$, and over $\frac{1}{3}$; women $\frac{1}{3}$, $\frac{2}{3}$, and $\frac{2}{3}$; and the total average increase

in each case was 25, 22, 21.8, 18 $\frac{1}{2}$, 14 $\frac{1}{2}$, and 14 pulsations in their order. The hour of minimum day pulsation was midday in one half of the whole observations, and then 11 a.m. and 1 p.m. each in one sixth of the observations.

Maximum pulsation in the working day.—It has already been stated that there were commonly four maxima, which, as they depend upon food, may be designated as breakfast, dinner, tea, and supper maxima.

1st. *Breakfast maximum.*—The average pulsation immediately after breakfast was 95, and was the lowest in females. The extremes were 76 and 113; and so great a variation as 20 pulsations has been noted in the same case in different days. The hour varied from 9 to 12 a.m., but was 10 in more than a majority of instances.

2d. *Dinner maximum.*—This was precisely the same in both sexes, and as the average pulsation was lower in females, the maximum is proportionately higher in that sex. The hour varied from 1 to 5 p.m., and was 3 p.m. in nearly one half, and 2 p.m. in nearly one third of the observations.

3d. *Tea maximum.*—The total average was 96 pulsations, or a medium between that of breakfast and dinner. It was lower in women than in men. The extremes were 81 and 108, and were somewhat less in each case than in the two former maxima. This maximum was not invariably present. The hour was from 5 to 9 p.m., but 7 p.m. was the maximum hour in one half of the observations, and then it was 6 p.m.

4th. *Supper maximum.*—This was not only the least of the maxima, but was the least constant, since it was found in only one half of the observations. It was less in females, as were also the breakfast and tea maxima. The extremes were 80 and 100, but the variation was inconsiderable in each case. The hour varied from 9 to 11 p.m., but was commonly 9 p.m.

Thus the four maxima were, on the average, 95, 97, 96, and 92, in their order, showing a difference of only 5 pulsations in the average maxima of 13 hours. The dinner maximum was the highest on the average, but not in every instance.

It was commonly so in the females, but each case varied, as we know each person to vary in the relative enjoyment of meals.

In reference to the foregoing part of the paper, we may sum up the sexual differences in the following sentences:—The women had lower pulsations, with less range and extremes at night; also more limited day range, with greater proportionate increase from the night. They had a little less increase in the morning ascent, and greater decrease in the evening descent. In them also the supper maximum was less frequent.

The following comprehensive table exhibits all the information obtained in reference to maximum and minimum pulsation and range of pulsation in each of the cases, and on every day of the inquiry.

No. II.—*Showing the maxima and minima Pulsation in the day and night, and the range of Pulsation at four periods in each case on each day.*

MEN.

CASE No. 73.

Lowest Pulse.				Highest in the Day.										Range of Pulsation.					
Night.		Day.		Breakfast.		Dinner.		Tea.		Supper.		Ex- treme.	1 to 5 a. m.		5 to 10 a. m.		10 a. m. to 5 p. m.		
Hour.	Pulse.	Hour.	Pulse.	Hour.	Pulse.	Hour.	Pulse.	Hour.	Pulse.	Hour.	Pulse.		Hour.	Pulse.	Hour.	Pulse.	Hour.	Pulse.	
Monday	7 a.m.	76	12, 1	99	10, 11	106	4	110	7	108	10	100	24	18	26	12	22		
Tuesday	6	75	12, 2	100	10	113	3	120	6, 7	108	—	—	45	5	38	10	20		
Wednesday	5	81	12, 4, 5	100	10	111	1	104	6, 7	104	9, 10	104	30	9	30	10	13		
Thursday	6	74	2	99	10	107	3	106	7	106	—	—	33	18	33	10	16		
Friday	5	76	1	91	7, 10	103	2, 3, 4	96	6	103	8	99	27	10	27	10	13		
Saturday	3, 5	74 76	—	—	6, 9	92 88	11, 4	97 92	7	100	—	—	24	14	16	—	16		
Average													30.5	12.3	28.3	10.4	16.6		

CASE No. 80.

Monday	5	60 } 65 }	11, 1	83 } 84 }	10, 12	88 } 91 }	3	105	6, 7, 8	90	—	—	45	9	23	20	25
Tuesday	5	63 } 60 }	12	81 } 82 }	10	94	3	100	7	94	10	90	37	7	31	30	19
Wednesday	1, 3	62 } 66 }	12	82 } 71 }	10	91	3	94	5	98	9	98	38	14	26	32	21
Thursday	1, 5	68 } 56 }	10, 11	72 } 65 }	7, 12	87 }	2	84	7	88	—	—	22	9	12	16	17
Friday	4, 5	56 }	12	65 }	7, 9	82 }	4	100	7	95	9	80	44	10	26	20	35
Saturday	1, 5	60 }	2	76 } 80 }	4, 7	86 } 80 }	10, 11 1	86 } 87 }	3, 4, 5	88 }	9	84	45	32	26	—	13
Average																	
													38.5	13.5	24	23.6	21.6

CASE No. 88.

Monday	6	64 } 64 }	1	80 } 86 }	10	88 } 108 }	3	84	7	94	9, 10	88	30	16	24	22	15
Tuesday	7	64 } 66 }	1	86 } 84 }	10	100	3	103	6	100	9	92	44	2	44	13	22
Wednesday	4, 5, 6	60 } 64 }	12	84 } 84 }	9	94	2	98	5, 6	96	—	—	34	13	34	20	16
Thursday	6	60 } 64 }	11, 12	84 } 79 }	10	94	3	94	5, 6, 7	88 }	10	84	34	15	34	18	14
Friday	2, 4, 5	65 }	12	79 } 96 }	7, 9	88 } 96 }	2	88	6, 8	89 }	—	—	32	4	31	17	18
Saturday	1, 4	62 }	4	79 } 88 }	7, 9, 10	88 } 94 }	3, 5	89 } 88 }	—	—	9	100	38	6	30	—	21
Average																	
													35.3	9.3	32.8	18	17.6
Total average													34.7	11.7	28.3	17.5	18.6

Table showing the maxima and minima Pulsation in the day and night, and the range of Pulsation at four periods in each case on each day.

WOMEN.

CASE No. 33.

Lowest Pulse.				Highest in the Day.								Range of Pulsation.					
Night.		Day.		Breakfast.		Dinner.		Tea.		Supper.		Range of Pulsation.					
Hour.	Pulse.	Hour.	Pulse.	Hour.	Pulse.	Hour.	Pulse.	Hour.	Pulse.	Hour.	Pulse.	Ex- treme.	5 a. m.	10 a. m.	5 to 10 a. m.	9 p. m.	19 a. m.
	52	12	75	10, 11	76	1, 3	83 84		7	84	10, 11	80	32	12	19	25	12
Monday . . . 4 a.m.	55 56	11, 12	78	10	79	2, 3	88 89		6	92	10, 11	84	37	6	23	28	16
Tuesday . . . 1, 5, 6																	
Wednesday . . . 3, 4	54	10, 12	74 76	9, 11	76 77	3, 5	84 86		8	84	—	—	32	2	20	18	12
Thursday . . . 5	55	4	76	—	—	3	84		6	86	—	—	34	7	19	23	18
Friday . . . 6	54	1	77	11, 12	80	2, 3	88		6	81	—	—	34	1	22	13	16
Saturday . . . 2, 4	54	3	79	6, 11	78 85	5	94		—	—	—	—	40	8	16	—	20
Average											6	19.8	21.4	34.8	15.6		

CASE No. 34.

Monday	5	71	11	85½	10	91	3	99	7	98	—	28	17	20	12	14
Tuesday	5	72	11	84½	9	94	2	100	8	92	—	28	6	22	16	16
Wednesday	5	69	12	89	10	100	3, 5, 6	99	8	98	10	88	31	5	31	9
Thursday	7	72	12	83	10	102	2	98	8	100	—	30	5	30	16	19
Friday	6	65	12	82	8, 9, 11	92	2	96	7	92	9	92	31	6	27	14
		70		89												
Saturday	2, 6	72	2	82	10	99	5	102	6, 7, 8	102	—	32	8	27	—	20
Average																
													30	7.9	26.1	14.4
																15.6

CASE No. 35.

Monday	1	62	5	88	—	—	3	98	—	—	9	100	38	10	21	25	15
Tuesday	3, 5	65	11, 3	92	10	97	1, 2, 4	104	7	104	—	—	39	10	32	28	12
Wednesday	2	65	12	93	10	100	3	101	6, 7	100	10	98	36	8	27	28	11
Thursday	3	67	11, 12	90	10	102	2	98	8	100	—	—	35	8	26	29	10
Friday	2	68	12	88	8	98	2	104	6	98	9, 10	96	36	0	25	8	16
Saturday	3	66	1	92	10, 11	104	5	108	9	100	—	—	42	22	36	—	16
Average																	
													37.6	11.1	27.8	23.6	13.3
Total average																	
													34.1	8.3	24.5	19.8	14.8

B. DISTURBING INFLUENCES.

These are sleep, food, sunlight, exertion and excitement, temperature and humidity; and of these the three former had the greatest, most constant, and continued power in these investigations.


1. *Sleep.*

This is represented in diagrams Nos. 1 and 2.

It causes a depression of the pulse, whether by night or day; but that depression is vastly greater which accompanies night sleep. Hence sleep is not necessarily associated with the lowest state of pulse, but it is probable that the soundest sleep is at night, when the pulse is low, and that sound sleep cannot occur with the elevated pulse of day. This is probably dependent upon the presence and absence of sunlight, as will be proved presently.

Waking out of sleep by night or day causes an immediate elevation of the pulse, and a permanent one so long as wakefulness continues.

Sleep at night occurs after the pulse has began to fall and whilst it is yet falling; when it is at its minimum; and, lastly, whilst it rises to constitute the morning elevation. As the morning rise is not due to sleep, so it is probable the evening fall is partly due to other influences; or since the minimum pulsation does not occur until the second, third, or fourth hour of sleep, the effect of sleep must be accumulative. But there is no accumulation in a second or third hour of sleep during the day, and therefore probably none during the night. Hence it is inferred that there are other causes than sleep producing the night fall of pulsation, and, consequently, that the whole night fall is not a measure of the influence of sleep. Neither, perhaps, is the day fall from day sleep a measure of the full effects of sleep under possibly more favorable circumstances as the natural ones of night and darkness. Hence, again, the difficulty of deter-



mining the exact influence of sleep. When sleep occurs with a falling pulse, but before the minimum pulsation has occurred at night, it causes immediately a much greater fall—one quite disproportionate to the fall before sleep; but at the middle of the night, or in the early morning, when the minimum has occurred, no such disproportionate influence is exerted if the person fall asleep from waking. Sleep, therefore, helps to drag the pulse down in the early night, to keep it down, and to prevent the morning rise.

Thus it appears to be the most powerful soon after retiring to rest; but since sleep lowers pulsation, and is believed to be the most profound at night when the pulse is much lower than during the day, it is probable that the lower the pulse during sleep, the more profound is the sleep. This lowest pulsation does not correspond with either the period of noisy or of quiet sleep, for in some cases the sleep will be as noisy or as quiet (as the case may be) in the morning at 7 o'clock, with a rising and increased pulse, as it was at 1 to 3 a.m. with the minimum pulsation. I believe both the latter indications to be fallacious, but if they are valuable they would lead me to believe that in the cases under examination the relative soundness of sleep was not that of relative depth of pulsation. The depth of pulsation, and therefore of sleep also, would depend upon the hour and the nature of the last meal.

As the women had an earlier and a deeper subsidence of the pulse than the men; it may be inferred that they had sounder sleep, and at an earlier period of the night.

Measure of sleep.—The following is the measure as observed during the day in thirteen instances:—Fell 0, 4, 8, 8, 11, 6, 10, 10, 9, 7 = 7·3 medium; but on two occasions it rose 3 and 2 in the second hour; and once it rose 4 in the first hour, and fell 9 in the second hour. This rise, if deducted from the fall, would leave an average of 5 pulsations fall from sleep. This is sleeping under difficulties, but even then I am of opinion that the common subsidence of the pulse would be from 8 to 10 pulsations.

In the night the effect of sleep is doubtless greater. In

one instance, at 4 a.m., the pulse fell from waking to sleep 18 pulsations ; but, as above stated, it varied with the period of the night. There were also numerous exceptions to the rule that the pulse falls with sleep and rises on waking ; but unless we could measure the co-ordinate influences, we cannot determine the effect of sleep alone in the night. I believe it may be reasonably estimated at 10 pulsations, with the understanding that it is greater before the minimum pulsation, and less at and after the minimum. The effect of the first hour's sleep is no exact measure. It was very various, as follows :—12, 6, 8, 6, 4, 6, 14, 10, 16, 6, 12, 2, 4, 6, 3, 10, 13, 20, 4, 8, 12, 8, 12, 8, 4, 7, 10, 18, 8, and 4 ; and in a few instances there was no fall. The effect of waking was commonly to raise the pulse 8 to 10 pulsations.

This interesting inquiry may be pursued to any extent by the aid of Diagrams No. 1 and 2, due allowance being made for occasional errors in determining which was sleep and which was not sleep in the quiet repose of the night, and for coughing, involuntary attempts to repress the cough, and for mental emotions, all of which tend to raise the pulse.

2. *Effect of Food.*

The food and degree of its enjoyment are important to this inquiry, and were as follows :

Breakfast.—No. 88, egg, bread, butter, and tea ; No. 80, the same, but coffee instead of tea, and bacon on June 11th ; Nos. 73 and 34 as No. 80 ; No. 35, the same, without egg.

Dinner.—All took meat, bread, and rice or potatoes ; Nos. 88 and 34 had porter ; and Nos. 35, 73, and 80 had ale. No. 80 had only boiled rice on June 14th.

Tea.—All had bread, butter, and tea, except No. 34, who had milk. No. 73 had always an egg ; and No. 80 had egg on June 13, 14, 15, and 16.

Supper.—No. 88 had cold meat, bread, and porter ; No. 80, meat, bread, and water or milk ; No. 73 had milk ; and Nos. 34 and 35 rice and milk.

All ate heartily and enjoyed the food, except No. 88,



who took a light tea on June 11, 12, 13, and 14, and had only a moderate dinner on June 16. No. 80 had a bad dinner on June 14, and a moderate one only on the 16th. No. 73 had a small breakfast and dinner on June 16. No. 34 had two ounces of wine at about 11 a.m. daily.

The effect of food is almost uniformly to cause a temporary elevation of pulsation, so that the pulse is more frequent soon after a meal, and subsides before the forthcoming meal. Thus there are commonly as many elevations and depressions of the pulse as there are meals. The meal hours were $8\frac{1}{2}$ a. m., $12\frac{1}{2}$ p. m. ($12\frac{3}{4}$ for the men), and 5 p. m., and a fourth at $8\frac{1}{2}$ p. m., not necessarily provided by the hospital, but yet, in these investigations, always obtained. The exceptions were as follows: 1st, on the first day the men took breakfast after 9 a. m.; 2d, Nos. 73 and 80 took dinner after 1 p.m. on the first three days, and No. 88 on the first two days; 3d, on the fifth day the dinner was deferred three hours. These caused a difference in the hour of elevation, for the elevations depend chiefly upon the hour and number of meals. With the above exceptions the examinations were made almost immediately after the conclusion of the meal.

The following table contains the amount of the elevations due to each meal, and also the hour of highest pulsation after the meal in each case, and on each of the six days' examination.

TABLE NO. III.—Showing the extreme amount of increased Pulsation after Meals, from the Hour before the Meal and the Hour after the Meal at which the Pulsation was the highest.

MALES.

First Day.			Second Day.		Third Day.		Fourth Day.		Fifth Day.		Sixth Day.		Average increase.
Meal.	Increase of Pulse.	Hour of greatest increase.	Increase of Pulse.	Hour of greatest increase.	Increase of Pulse.	Hour of greatest increase.	Increase of Pulse.	Hour of greatest increase.	Increase of Pulse.	Hour of greatest increase.	Increase of Pulse.	Hour of greatest increase.	
73	Breakfast .	18	1st, 2nd	36	2nd	29	31	2nd	7	2nd	19	1st, 3rd	23.3
80	"	11	1, 3	30	2	26	{ decrease diarrhea }		13	1	8	2, 3	15.6
88	"	9	1	34	2	24		23	1	1	1	16	1, 2
73	Dinner . .	11	2, 3	20	2	0	2	3	5	2, 3, 4	2	1	8
80	"	21	2	18	2	11	4	2	35	4	0		18
88	"	4	2	17	2	14	10	3	9	2	{ sleep }		11
73	Tea	8	2	8	1, 2	4	4	2	11	1		14	2
80	"	8	1, 2, 3	10	2	{ 8 } decr.	20	2	11	2	0		
88	"	12	2	12	1		0	0		9	1, 3	0	
73	Supper . .	2		0		2	0		0		0		
80	"	0		{ decrease }	2	2	0		0		6	1	
88	"	6	1, 3		4	1	0	4	2	0		10	1

FEMALES.

35	Breakfast .	7	3, 4	8	2	17	5	1	0		20	2, 3	16.4
34	"	17	2	13	1	11	26	2	0		17	2	16.8
33	"	6	2, 3	15	2	10	15	4	8	3, 4	23	3	12.8
35	Dinner . .	10	3	7	1, 2	8	10	2	16	2	8	2	10
34	"	5	3	12	2	11	15	2	14	2	26	1, 2	11.5
33	"	9	1, 3	11	2, 3	8	11	3	8	2, 3	15	2	9.5
35	Tea	8	1, 2	8	2	12	6	1	2	1	0		
34	"	12	2	0	0	0	12	3	8	2	0		
33	"	4	2	4	1	0	4	1	1	1	0		
35	Supper . .	2	1	0	0	6	12	1	5	1, 2	4	1	
34	"	0		0	0	0	0		3	1	0		
33	"	4	2, 3	4	2	0	0		0		0		

Hour of Highest Elevation after a Meal.

As the examination was not made at the end of an hour after the meal, but within half or three quarters of an hour after the commencement of the meal, it must be understood that the term means "during an hour." The maximum hour varied from the first to the fourth, but the latter was too infrequent to need observation. The second hour was as frequent as both the first and third hours combined; but there was almost always an increase during the first hour, and so frequently that that hour was second only to the second hour. Thus the order of frequency of the maximum hours were the second, first, third, and fourth. In many instances the highest pulsation occupied two or even more hours. The first was most frequently the maximum hour after tea, supper, and breakfast, in their order; whilst after dinner it occurred in only four out of thirty-seven observations. The first hour was much more frequent after breakfast, and also (but in a less degree) after supper in men; and the third hour after breakfast and dinner in women.

Amount of the highest hourly Pulsation.

It is difficult to isolate the effect of the meals from that of exercise and temperature; but with the exception of the breakfast, we shall not go far wrong in computing the elevation from the returns of the preceding hour. This rule would, however, be wrong for the breakfast, since the elevation is often then so great as 36 pulsations in the men, and 25 in the women; and there are other elements of error then operating, as those of awaking from sleep and of rising.

1. *Breakfast.*—I endeavoured to isolate this influence on the fifth and sixth day, by directing all the patients to rise at 6 a.m. instead of 8 a.m., and to take a walk out in the intervals of the examinations. The three men and two of the women did so on the fifth, and on the sixth morning

two men and one woman also. On the fifth morning the highest increase from the hour before breakfast was 13, 7, and 1 pulsations in the three men, and 0 and 8 in two women. On the sixth day, it was 16 and 8 in the men. Thus the average pulsation due to breakfast is probably from 8 to 10 beats per minute; but the average increase observed, during the six days, from the hour preceding the breakfast, was 23·3, 15·6, 17·8 in the men, and 11·4, 16·8, and 12·8 in the women, or a total average of over 16 pulsations per minute.

2. *Dinner*.—This varied from nothing to 35 pulsations. On the average it was—men, 8, 18, 11 = 12·3 medium; women, 10, 11½, 9½ = 10 medium, or a total average of 11 pulsations. Thus it was less in women, and in them also the variations were less.

3. *Tea*.—The average for the first few days was—men, 7, 11, 7 = 8·3 medium; women, 7, 6, 3 = 5·3 medium; and the total average was about 7 pulsations. The variations were less than at either of the preceding meals; viz., 1 to 20 pulsations, and they were nearly equal in the two sexes.

4. *Supper*.—In eleven instances in the women, and nine in the men it was null. The average increase was—men, $\frac{3}{3}$, less than 2, 4 = 2½ medium; women, 5, ½, 1½ = 2½ medium. The variations were 0 to 12 pulsations. The sexes were about equally wanting in uniformity.

Thus the average increase at each of the meals was—breakfast 8 to 10 (estimated), dinner 11, tea 7, supper 2½ pulsations.

The proportion to the whole number of meals in which no increase was observed was as follows: breakfast—men 0, women $\frac{5}{36}$; dinner—men $\frac{1}{36}$, women 0; tea—men $\frac{3}{36}$, women $\frac{9}{10}$; supper—men $\frac{9}{36}$, women $\frac{11}{36}$.

In the women the increase was commonly less, but more constant, than in the men, as was also the longer period before the maximum was attained after breakfast and dinner.

In this part of the investigations two other inquiries were made; viz., the effect of early rising and of fasting.



The effect of Early Rising.

This is shown in the returns of the fifth and sixth day, in cases 80 and 88 ; and on the fifth day in cases 73, 33, and 34, all of whom then rose directly after 6 a.m., and walked round the hospital quickly between 6 and 7 a.m., and again between 7 and 8 a.m. Case 35 also rose at 7 a.m. on the fifth day, and walked out between 7 and 8 a.m. The constant effect was to produce an unusual increase of pulsation at the breakfast hour, and more particularly at the first hour after rising. In the men, the increase was confined to the first hour, and was, on the fifth day, 18, 18, 12 ; and on the sixth day, 24 and 18, or an average increase of 18 pulsations per minute. In the second hour no further increase took place ; but, on the contrary, a decrease on the fifth day of 5, 5, and 1, and the sixth day of 10 and 2, or an average of 4.6 pulsations. The increase was less on the fifth than on the sixth day, because then the patients were awake at the 6 a.m. examination, and the pulse had consequently risen much. In the women, the increase on the fifth day was—first hour, 23 and 33, second hour, 4 and 2 pulsations. This increase was much greater than that of the men ; but it may be explained by stating that they had literally interpreted my directions, “to take a run round the hospital.” Case 35, who rose at 7 instead of 8 a.m., and walked round the hospital, had an increase of 5 pulsations.

But the numbers now given are not entirely due to the early rising, for the pulse would have risen somewhat at that period of the morning had they remained in bed.

The excess of pulsation over the average at these hours is that due to the early rising, and was, on the total average, 10 pulsations per minute. This large and unusual amount of increase from the early rising was followed by an unusual decrease of pulsation, and hence it is desirable to know the effect of early rising on the pulsation on the first and second halves of the day, and also on the whole day. The effect

during the morning was to increase the pulsation by an average of about 4 pulsations per minute. The increase was more than twice as great in men as in women; viz., 5, 6, men, and 2, 4, women; and consequently in the latter there was greater reaction. The pulsation in the latter half of the day showed a decrease to the extent of 4, 1, in the men, and 2, 3, in the women. The effect upon the total pulsation of the whole day was a slight diminution in the rate; and, upon the whole, the effect of occasional early rising was to cause an earlier and a deeper subsidence of the pulse at night, and consequently earlier and deeper sleep.

3. *Relation of hourly temperature and humidity to pulsation.*

The returns are restricted to those obtained within the two wards of the hospital in which the males and females severally lived. That of the men's ward only was taken with both the dry and wet bulb thermometers, so that that alone will enable us to determine the degree of humidity of the air.

The progress of pulsation and temperature is shown in the following average table for each day.

TABLE No. IV.

	Temperature. Men's.	Pulsation.		Temperature. Women's.
		Men's.	Women's.	
Monday	—	86·2	80·6	66·5
Tuesday	—	86·5	80·8	65·6
Wednesday . . .	67·1	86·9	82·2	66·1
Thursday	65·6	82·5	81·2	64·7
Friday	64·5	82·9	80·0	63·6
Saturday	63·7	81·2	83·6	62·5

Thus the temperature declined on each day, except the third, in the women's ward; and the pulsations of the men declined also.

The extreme daily range in the hourly averages of the six days was only 1.3° in the women's, and 2.2° in the men's ward. The maximum in the women's ward was attained at 4, 5, or 6 p.m., and thenceforward the temperature declined through the night to 6 and 7 a.m. From 9 a.m. to the maximum, the increase was only 1° , and the temperature at midday was only 2° above the minimum. In the men's ward the maximum was about midnight, and the minimum at 10 or 11 a.m. Thus the lines of temperature and pulsation so far agree, that they both decrease through the night, and increase through the day, but the maximum and minimum hours differ. Moreover, at night, the pulse falls whilst the temperature is at its highest; in the morning, it rises whilst the temperature falls to its lowest; and through the day, from 9 a.m. to 9 p.m., maintains a somewhat even relation, whilst the temperature attains its minimum, increases through the day, and nearly attains its maximum at night.

The temperature varied from hour to hour, as did the pulsation; but the line of temperature in each of the six days, when compared with the pulsation of those days, offers no instance in which the pulse appeared to be directly under the influence of hourly changes of temperature. The average hourly changes are shown in Diagram No. 4.

Humidity.

The interest of this inquiry is its relation to the warming of hospitals, for I have elsewhere proved that with elevation of seasonal temperature there is commonly increased dryness, and with both these conditions are increased pulsation and lessened rate of respiration—two conditions not tending to health.

The difference between the dry and wet bulb thermometers in the external air on the six days was 13.1° , 9.9° , 2.9° , 4.2° , 4.6° , and 2.7° , showing a very great variation in one week; whilst the mean difference in the hospital was so uniform on the four last days as 6.2° , 6.4° , 6.2° , and 6.6° . The

average difference on these four days between the dryness of the external and internal air was, external 3.6° ; internal, 6.5° ; and, consequently, on these days the internal air was twice as dry as the external; rain fell externally on these days, and thus the humidity would be greater.

The following table gives the average hourly returns for four days of the difference between the wet and dry bulb temperature :

TABLE No. V.

Hourly average difference between the wet and dry bulb Thermometers.

	a.m.											p.m.												
Hour . .	12	1	2	3	4	5	6	7	8	9	10	11	12	1	2	3	4	5	6	7	8	9	10	11
Degrees .	6.7	6.5	6.6	6.9	6.2	6.7	6.6	6.6	6.7	6.5	6.3	5.9	6.1	6.2	6.8	7.4	6.5	5.6	6.1	6.6	6.6	6.5	6.6	6.4

The uniformity in the amount of humidity, as indicated by the difference between the dry and wet bulb temperatures, is very remarkable. There was a little average increase at 11 a.m. and 5 p.m., with a slight decrease at 3 p.m. Thus tolerable uniformity was attained; but the dryness was too great as compared with the external air, but equal to that for the whole of the same quarter of the year.

4. Influence of Exercise and Excitement.

The returns of the morning of early rising prove clearly that the pulse may be increased at an unwonted hour by exercise, but from the fact that, at 10 a.m., the pulse may attain to the same height without the patient having risen that it would have attained had he taken exercise during each of several preceding hours it is evident that the maximum pulsation of the day was uninfluenced by it. The effect of violent exercise is great but temporary, and if the

lying posture be maintained for five or ten minutes after ordinary exercise no trace of its effect remains. Ordinary and quiet exercise had but very little influence over hourly pulsation.

5. *Effect of Sunlight.*

It has been proved that between the pulsation of the night and the day there is a difference of 25 to 40 pulsations and upwards per minute, and we have proved that sleep alone does not reduce the pulse to its great depth at night, nor food alone raise it to its great height during the day. Moreover hourly temperature has no influence that can be defined, and moderate exercise is powerless, and hence we seek for other powerful causes for this great difference. The day has light and the night the absence of it, and it will be seen that the highest pulsations occurred (in June) at the period of the day when the daylight was most powerful, that both began to increase together in the morning and to decline together in the evening. Thus sunlight is a probable cause; but I have not been able to prove it absolutely by inducing a consumptive patient to remain in absolute darkness through the day, and in the winter season the test would be insufficient. This influence may be estimated in three modes: 1st, by subtracting from the difference between the highest pulsation of the day and the lowest of the night the influence of food and of profound sleep; 2d, by taking, in like manner, the difference between the lowest pulsation of the night and the highest after the breakfast and deducting the influence of breakfast and of light sleep; and, 3d, by taking the difference between the pulsation at 4 a.m. when it was light and mid-day, allowing a little for light sleep, and for the remaining influence of breakfast. Either would represent the influence if we could estimate each of the quantities concerned in the calculation with accuracy.

We will now investigate this subject in the three directions just indicated, and allow, according to the preceding

inquiries, 10 pulsations for profound sleep, 10 for breakfast, 11 for dinner, and 5 each for light sleep, and for the remains of the breakfast influence at mid-day.

The average difference between the highest and lowest pulsations were 24, 29, 28·7, 30·2, 22·5, and 30 = medium, 27·4. The average difference between the lowest at night and the highest after breakfast was, 24, 20·2, 30·3, 24·3, 22·1, 26·6 = 24·5 medium. The average difference between the pulsation at 4 a.m. and mid-day was 15·2, 9·9, 20·6, 22·5, 11·7, and 19·4 = 16·5 medium. The calculations will, then, finally be—

$$\left. \begin{array}{l} 27\cdot4, \text{ less Dinner } 11 \text{ and Sleep } 10 = 6\cdot4 \\ 16\cdot5, \text{ less } \frac{1}{2} \text{ Breakfast } 5, \frac{1}{2} \text{ Sleep } 5 = 6\cdot4 \\ 24\cdot5, \text{ less } \frac{1}{2} \text{ Sleep } 5, \text{ Breakfast } 10 = 9\cdot4 \end{array} \right\} \begin{array}{l} \text{Medium } 7\cdot4 \\ \text{pulsations.} \end{array}$$

It thus appears that in consumptive patients in the month of June the sunlight exerts an influence amounting to from 6 to 10 pulsations average upon the pulsation of the day; and consequently its absence permits a corresponding reduction in the pulsation of the night. The effect in individual cases is oftentimes much greater.

PART II.

RESPIRATION.

HOURLY CHANGES IN THE RATE OF RESPIRATION IN PHTHISIS.

These have been determined by the investigation already described, and also by a supplementary one made upon 29 patients on the night of November 14-15, by the kind help of Mr. V. Edwards and Mr. Vise, our talented and esteemed Resident Surgeon and Clinical Assistant respectively,

(See diagrams, Nos. 1, 2, and 4.) In estimating the value of a relative change in the rate of respiration and pulsation the normal difference in the rate of the two functions must be remembered, and hence, that an increase of one respiration may be proportionately as great as of four pulsations.

The total average rate for both sexes exhibits a daily variation of 8 respirations with average extremes of $21\frac{1}{2}$ and $29\frac{1}{2}$, and absolute extremes of 13 and 49 respirations per minute. The minimum was at 8 a.m., and the maximum at midnight.

The hourly average changes are shown in the next table :

TABLE No. VI.

	a.m.											
Hour	12	1	2	3	4	5	6	7	8	9	10	11
Total Average Respirations .	29.5	28.3	27.8	25.9	26.3	35.6	25.1	23.6	21.5	23.6	24.1	24.5
Average Males	27.6	26.4	25.5	23.9	23.4	22.8	23.6	21.6	18.8	21.3	22.0	22.9
Average Females	31.4	30.3	30.4	29.0	29.3	25.7	26.6	25.5	24.2	25.4	26.3	26.3
	p.m.											
Hour	12	1	2	3	4	5	6	7	8	9	10	11
Total Average Respirations .	24.7	25.8	24.7	25.8	24.7	25.5	26.4	26.3	25.4	25.6	25.0	29.2
Average Males	23.3	23.1	23.1	24.8	24.6	24.8	23.5	24.0	23.7	23.5	23.5	29.2
Average Females	26.2	29.0	26.0	28.2	28.9	28.1	30.4	28.6	27.0	27.1	27.5	29.2

Thus, at 11 p.m. there was a sudden increase of 4.2 respirations or one sixth of the total number at 10 p.m., and then a slight increase to the maximum at midnight. Thence it declined, but remained very high at 1 and 2 a.m., and had a slight increase at 4 a.m. ; whilst at 7 a.m. and 8 a.m. the decrease was great and sudden. Thus the hour of sudden increase corresponds with that of the commencement of sleep, and of sudden decrease with that of permanent waking. At 9 a.m. it had increased 2 respirations

from the minimum (being then the same as it was at 7 a.m.), and thence increased progressively (with a disproportionate increase at 1 p.m.) through 3 respirations to 6 p.m., and finally decreased until 10 p.m., when the sudden increase from sleep occurred.

Thus the decreasing hours were 7 to 10 p.m. and 3 to 8 a.m. (minimum), whilst the increasing were 9 a.m. to 6 p.m. and 11 p.m. to about 2 a.m. (maximum). The hours of sudden decrease were 3 a.m. and 7 and 8 a.m., and of sudden increase 11 p.m., 9 a.m., and 1 p.m.

Thus as a general expression it may be stated that the respiration increases suddenly and greatly in the early night, decreases thence to the period of rising, increases gently and progressively through the working day until after the tea hour (5 p.m.), and finally falls to and after going to bed.

The rate is greater during the night than the day. Thus in intervals of eight hours, as 11 p.m. to 6 a.m. (night) and 3 p.m. to 10 p.m. (day), and also 9 a.m. to 4 p.m. (day), the average is 27·2 (night), 26 and 24½ (day). There are exceptions to this statement; but when those are included, the rule is as just laid down.

The difference in the average returns from the two sexes was but slight. Thus, in females, there was a greater rate, a less range (7·2), and, consequently, more uniformity of respiration; a higher maximum (31·4) and minimum (24·2) rate, with the maximum one hour earlier (viz., midnight instead of 1 a.m.); and a less increase at 11 p.m. There was also a less proportionate increase after breakfast, with a greater increase at 1 and 6 p.m., and, consequently, a more tardy fall in the evening.

Influence of Sleep.

This is in general very great and decided, and tends to increase the rate of respiration. It may be estimated in two ways—1st, by contrasting the rate in successive hours, when the patients are alternately awake and asleep; and

2d, by ascertaining the effect of sudden waking out of sleep.

The former is liable to the following fallacies: 1st. If the full effect is not immediate, it will be influenced by the state of waking or sleeping in the intervals of the examination, and of this state we have no knowledge. 2d. The effect of sleep is in relation to its intensity; and of the degree of intensity we have no precise measure; and hence a light sleep may exhibit a change not far removed from that of wakefulness; and awaking from a light sleep may scarcely alter the rate observed during that sleep. 3d. Involuntary attempts to prevent cough in sleep retard respiration, for a limited period at a time, and thereby lessen the respirations during sleep at the moment of examination; whilst the fit of coughing in which the patient often wakes increases the respirations, so that they are then beyond the normal rate of waking.

A. *Waking from Sleep.*

In the examinations detailed in Diagrams Nos. 1 and 2, I usually found that this effect was to lower the rate from 5 to 8 respirations per minute. In the examination on November 14-15, the effect was precisely noted in fourteen instances, and recorded. In two instances no change occurred (14 and 21 per minute), but in the remaining twelve, the decrease with waking was as follows: 18, 12, 10, 7, 6, 6, 5, $4\frac{1}{2}$, 3, 3, $2\frac{1}{2}$, and $1=6\cdot5$ medium.

To this may probably be added some unknown quantity, for it is highly improbable that the whole decrease took place in one minute, judging from what I have seen in health. No observation was made at a later hour than 3 a.m., for after that the patients either remained asleep during the whole examination, or were fully awake at the examination; but there is reason to believe that the decrease referred to is greatest in the early part of the night. It is also certain that, when the respirations are not much beyond 20 per minute, the decrease on awaking is small, and that so great

a decrease as 18 or even 10 respirations cannot occur with that rate of respiration ; but it does not appear that the decrease is altogether proportioned to the frequency of the act, except when conjoined with depth of sleep.

B. Hourly changes from Sleep to Wakefulness.

There was much variation in the returns, and especially in the first night, when the novelty of the examination disturbed the rest of the patients ; but the rule is that the respirations are more frequent in an examination during sleep than during wakefulness.

The following are instances supporting the rule :

Diagram No. 1.—June 11. 4, 7, and 8 a.m. ; 4 and 11 p.m. June 12. 4 and 8 a.m. ; 11 p.m. June 13. 1, 4, 6, 7, and 8 a.m. ; 4, 11, and 12 p.m. June 14. 1, 5, 6, 7, 8, and 11 a.m. ; 2 and 11 p.m. June 15. 2, 4, and 7 a.m.

Diagram No. 2.—June 11. 1, 2, and 7 a.m. June 12. 1, 2, 3, 4, and 8 a.m. June 13. 6 a.m. ; 11 and 12 p.m. June 14. 7 a.m. ; 11 and 12 p.m. June 15. 4, 5, and 7 a.m. June 16. 5, 6, and 8 a.m.

The amount of the increase observed at the hour of sleep, above the respiration at the hour of waking, varied very greatly. In Case 45, it was once 15 ; Case 43, 17 (two succeeding hours) ; Case 15, $17\frac{1}{2}$ and 15 ; but when observed twice in the same case, it was never precisely the same. It was often very great in Cases Nos. 35, 73, and 80 (Diagrams 1 and 2). In Case 80, the increase during the first hour of sleep was very striking, and in the six days was as follows : 8, 15, 12, 10, $9\frac{1}{2}$, and $12=11$ respirations medium. In Case 35 (woman), it was also 11, 9, and $12=10\cdot6$ medium.

The highest rate of respiration commonly occurred in the early part of the night, and the respirations were fewer as the morning advanced, but in some instances, after the subsidence at 2, 3, or 4 a.m., there was another marked increase at 5, 6, or 7 a.m., corresponding with what is

known popularly as second sleep. So long as sleep in any degree remained, the respirations were more frequent than in the hour of permanent waking; and in the instance of second sleep, in Case 34, the fall of respiration from the last hour of sleep to the first of permanent waking was, in the six days, so great as 12, 11, 18, 12, 12=13 respirations medium.

Influence of Food over hourly rate of Respiration.

Diagram No. 4 is available only for the women, since the meal hours for the men varied during the first two days. The meal hours for the women were 8½ a.m., 12½, 5½, and 8½ p.m. The following is a general description. The rate increased greatly directly after breakfast, and continued high (with or without a little further increase), until soon before dinner. Again, it suddenly increased directly after dinner, and as suddenly fell at 2 or 3 p.m., but increased again before the tea, and still further after that meal; and then finally sank rapidly, until sleep occurred. Thus it rose directly after each meal, and fell immediately before a meal, but in such a manner that the respiration before the dinner was higher than before the breakfast, and that before the tea higher than that before either of the preceding meals. The march may thus be represented by a series of platforms one higher than another, each one having ascending and descending steps. The lowest platform is before breakfast, and is gained by a descent from sleep; the second, after breakfast; the third, after dinner; the fourth, after tea, when the descent is great; and, finally, the fifth, during the sleep of the night.

The average increase from the meals was—breakfast, 1½; dinner, 3; and tea, 2½ respirations. The supper meal but rarely caused any increase. One case (35) had the greatest increase from dinner and tea (4 respirations), whilst it had no increase after breakfast; and the variations as to the

relative influence of meals in the same day was very great.

It is difficult to estimate the average duration of this influence, since the rate progresses throughout the day, but it is probable that it was the greatest after the breakfast, whilst the amount of increase was the greatest after dinner and tea.

Thus the following facts are established :

1. There was increased rate of respiration from food, and it occurred in the first hour, and passed away more or less in the second hour. The greatest want of uniformity was evident after supper, and the least after dinner and tea.

2. The extent varied greatly in each case, but was the greatest after dinner, tea, breakfast, and supper, in their order. The extremes held the same order, and were 1 and 10, 1 and 6, $\frac{1}{2}$ and 5, and 1 and $5\frac{1}{2}$ respectively.

3. The peculiarity of each case in reference to the effect of certain meals over others was remarkable. Thus, in 73, there was no effect from any dinner, and only once from the tea, whilst the effect of both breakfast and supper was considerable. On the contrary, Cases 33 and 35 had a largely increased rate from dinner and tea, and but a very small ~~one from~~ breakfast.

4. The sexual differences were chiefly—

1. Women had more uniformity, rapidity, and less duration of effect. The increase during the first hour was greater after the dinner and tea meals than after breakfast and supper, whilst a decrease in the first, and often in the second hour also, was common after supper.

2. Men had a larger increase after the breakfast, with a much more frequent decrease in the first, and ~~increase~~ in the second hour after dinner.

Effect of Early Rising upon the hourly rate of Respiration.

As sleep increases the respirations, and waking lessens them, so early rising lessens those of the night. The effect was varied in the different cases. Nos. 85, 88, and 80 obeyed the rule, whilst scarcely any difference was experienced by No. 34; and Nos. 33 and 80 had an increase during the first, and a decrease during the second hour. This is remarkable, since the effect upon the circulation was to increase it in all cases.

Since the breakfast hour was not changed with the early rising, the respirations were not only decreased, but kept low, and the minimum was attained somewhat earlier. The effect upon the later respirations of the day was to increase them between breakfast and dinner, in Cases 73, 33, and 88, and even until the tea hour, in Case 73; whilst in four cases, Nos. 33, 34, 80, and 88, they were lessened in the after half of the day. The effect upon the respirations of the whole day was too varied to be defined.

Cases Nos. 73 and 33 were unduly influenced by exercise and excitement, 35 by food and sleep, whilst 88 and 34 were less influenced by either agent.

Effect of hourly variations of Temperature upon Respiration.

I have elsewhere proved that the general daily or weekly effect of increasing temperature is to lessen the rate of respiration.

The patients lived in two temperatures, the external and the internal; and thence the influence of one might modify that of the other. I have no records of the hourly changes in the external temperature, but it had probably little or no effect in these investigations, for the respirations were the most frequent at night, when the temperature was low, or at the lowest, and the least frequent near to midday, when, in June, the temperature must have been considerable. There is great correspondence between the lines of internal

temperature and respiration (Diagram No. 4); but the subject is one of difficulty, since the hourly variations of temperature were but small, and the changes in respiration have been accounted for when discussing the influence of food and sleep.

The highest temperature in the wards was in the evening, and even so late as midnight. Thence it declined until 5 or 6 a.m., and remained very low until 10 or 11 a.m., and then it gradually increased until the maximum was attained at night. Thus the lines of respiration and temperature so far correspond that both are highest in the night and lowest in the morning; and thence both increase through the day. This is a sufficient correspondence to lead us at least to seek for a law of dependence, but there are three circumstances which militate against this conclusion: 1st. The hours of maximum and minimum temperature are not those of respiration. 2d. It is but very rarely that any increase in temperature is accompanied by an increase of respiration. 3d. Sudden and great changes in respiration are not accompanied by similar changes in temperature.

Effect of Sunlight.

This, although powerful upon pulsation, is null upon respiration. The effect is neither a direct nor an inverse one, since the respirations are the most frequent when there is no sunlight, and are not the most frequent when the sunlight is the most powerful. The respirations are the fewest at 8 a.m., when, in June, the sunlight is considerable; and they decline up to that hour whilst the sunlight is increasing. They also attain to a very high point in the evening, when the sunlight has declined; and the greatest and most sudden rise occurs at 11 p.m., when there is no sunlight.



Ratio of the Respiration to the Pulse in Phthisis.

This varies with nearly every hour of the day and night, but is the highest in the night, when the pulsation is the lowest and the respiration the highest.

The following table exhibits the hourly ratios in all the cases combined :

TABLE No. VII.

	a.m.											p.m.												
Hour . .	12	1	2	3	4	5	6	7	8	9	10	11	12	1	2	3	4	5	6	7	8	9	10	11
Ratio as 1 to	3·6	2·5	2·5	2·6	2·6	2·6	2·9	3·2	3·6	3·6	3·7	3·5	3·5	3·4	3·7	3·6	3·4	3·7	3·4	3·5	3·5	3·4	3·4	2·7

Thus the maximum was at 1 and 2 a.m., but practically it may be said to extend from 11 p.m. to 5 a.m., or the period of lowest pulsation and highest respiration. At 6 a.m., when sleep is departing, it was lowered, and continued so until 10 a.m., when it was only two thirds of the maximum. It is then at the minimum, as it is also at the hours 2 and 5 p.m. Immediately after each meal the ratio is lessened, from increased pulsation, but in an hour or two it is again increased, so that the ratio is lessened directly after each meal, and increased before each meal; but after the tea hour it does not fall; and at 11 p.m. it suddenly gains the maximum. The effect of sleep in the night is much greater than that of food, since it always raises the ratio to the maximum, by increasing respiration and lowering pulsation. The average ratio was not so low as 1 to 4, nor so high as 1 to 2; but the following table shows that one of the cases had a ratio as high as 1 to 4, and never lower than 1 to 2·3. The lowest ratio observed was 1 to 5·8, but in the early stages of the disease it is often lower than that. Thus, in a soldier, six feet two

inches in height, a patient of mine, now at the hospital, it was, on November 14th, only 1 to 8 in the lying posture.

TABLE No. VIII.

Exhibiting the Average Ratios in each of the cases, and the average in each sex.

		Hour, a.m.											
CASE.		12	1	2	3	4	5	6	7	8	9	10	11
No. 73	Ratio of 1 to	2.6	2.7	2.6	2.9	2.8	2.99	2.7	2.8	3.1	3.0	3.3	3.0
No. 80	"	2.99	2.8	3.0	3.1	3.5	3.2	3.5	4.1	4.8	4.6	4.7	4.4
No. 88	"	3.0	2.8	3.0	3.1	3.0	3.0	3.1	4.5	5.0	5.1	5.6	5.1
Men's Average		2.8	2.7	2.8	3.0	3.1	3.0	3.1	3.8	4.3	4.2	4.5	4.1
Women's Average		2.4	2.4	2.3	2.4	2.4	2.4	2.7	3.0	3.5	3.5	3.6	3.3
No. 33	Ratio of 1 to	1.6	1.4	1.5	1.4	1.5	1.5	1.8	1.8	1.9	2.09	2.0	2.1
No. 34	"	3.0	3.3	3.0	3.3	3.2	3.2	3.3	3.7	4.4	3.8	4.1	4.0
No. 35	"	2.7	2.7	2.5	2.7	2.5	2.5	3.0	3.5	4.4	4.9	4.9	4.9

		Hour, p.m.											
CASE.		12	1	2	3	4	5	6	7	8	9	10	11
No. 73	Ratio of 1 to	2.8	2.9	3.0	3.0	2.8	2.7	2.99	2.9	2.88	2.7	2.9	2.3
No. 80	"	4.4	4.4	4.4	4.8	4.5	4.2	4.5	5.0	4.6	4.5	4.4	3.4
No. 88	"	4.8	5.2	5.0	5.8	4.5	4.6	4.8	4.6	4.6	4.6	4.8	2.8
Men's Average		4.0	4.1	4.1	4.4	3.9	3.8	4.1	4.1	4.0	3.9	4.0	2.8
Women's Average		3.6	3.3	3.7	3.5	3.5	3.4	3.3	3.5	3.7	3.6	3.3	2.9
No. 33	Ratio of 1 to	2.0	1.98	2.3	2.1	2.0	2.1	1.93	1.9	2.0	1.98	2.0	1.8
No. 34	"	4.0	3.9	4.4	3.7	4.1	4.0	4.2	4.5	4.5	4.2	3.5	3.2
No. 35	"	4.98	4.1	4.5	4.8	4.4	4.3	3.9	4.3	4.7	4.7	4.5	3.8

Thus whilst the ratios in each case varied much, and were much higher at every hour in the women than in the men, they support the statements made from the total average. No. 34 had, however, a less proportionate night increase than any other, so that its maximum was 1 to 3.0, and its minimum 1 to 4.5. The maximum hour in all was from 11 p.m. to 3 a.m., and once extended even to 4 and 5 a.m., whilst the minimum was from 10 a.m. to 8 p.m.

The maximum varied from as 1 to 1·4, to as 1 to 3·0. In some instances the ratio somewhat increased directly after the meal.

Summary.

The following is a summary of the foregoing communication :

1. The rate of both pulsation and respiration varies with each hour of the day and night ; that of pulsation on the average of the day to 26, and of respiration to 8 per minute.

The rate of pulsation is the lowest in the "night" (1 to 5 a.m.), and the highest in the "day" (9 a.m. to 9 p.m.), and rises greatly in the "morning" (5 a.m. to 10 a.m.), and sinks greatly in the "evening" (9 p.m. to 1 a.m.) The rate of respiration is the greatest in the night, and the least at about 8 a.m. ; after which it increases until about 6 or 7 p.m., when it declines to the hour of sleep ; so that it is the least before breakfast, then before dinner, then before tea, and the highest when the patient is asleep at night.

2. The average variation of pulsation is greatest in the "morning" (27), then in the "evening" (19), "day" (7), and "night" (10).

3. The night minimum of pulsation (65) was most frequently at 5 a.m., and the day minimum (84) was 20 pulsations above it. The day maxima were (breakfast) 95, (dinner) 97, (tea) 96, and (supper) 92.

4. In women the pulsation was lower and more uniform, and with a greater night subsidence and less morning elevation. The respiration was quicker and more uniform, and the sudden increase at night was not so great, because the respiration did not fall so low after tea.

5. The day increase of pulsation is chiefly due to food and sunlight, but of respiration to food alone ; whilst the decreased night pulsation is due to darkness and sleep, and the increase of respiration to sleep only. The effect of sound sleep was to lower pulsation 8 to 10, and increase

respiration about 10 per minute; whilst light sleep may be estimated from one half of these numbers to nothing.

Thus during sound sleep the pulse falls greatly for two or three hours, and the respirations are at their highest; but as the morning advances, and whilst the patient is yet asleep, the pulse rises and the respiration falls. When second sleep occurs, at 4, 5, 6, or 7 a.m., the respirations are again increased, and then the fall on final waking averaged 13 respirations per minute. The pulse is not commonly depressed by the second sleep. The deeper the sleep, the more frequent the respirations; but the pulsation is not always then the lowest. The pulse falls and the respirations rise on sleeping, and both the contrary on waking out of sleep.

6. Food increases both pulsation and respiration, on the average, as follows: Breakfast 8 to 10 and $2\frac{1}{4}$; dinner 11 and 3; tea 7 and $1\frac{1}{2}$; and supper $2\frac{1}{2}$ pulsations. There is great variation in both functions.

The maximum effect was earlier in respiration than pulsation, and in the former was commonly within one hour, except after supper, and endured longest after breakfast; whilst in the latter (pulsation) it was chiefly the second hour, and was the second, first, third, and fourth hour in their order of frequency. In women the effect on respiration was more uniform, rapid, and evanescent.

7. Fasting (for a short time) caused more pulsation, and in one half of the cases more frequent respiration also.

8. Early rising increased pulsation before breakfast and before dinner, but lessened it in the after part of the day, and in the day taken as a whole, and it produced an earlier and a deeper sleep. It lessened greatly the respirations before breakfast, and sometimes increased those of the afternoon.

9. Hourly changes of temperature correspond more with respiration than pulsation, but probably are not causative in either function.

10. Moderate exercise has no appreciable effect on either function if the lying posture be maintained afterwards for a few minutes.

11. The effect of sunlight, in June, is to increase pulsation on an average of 6 to 10 per minute; but it has no influence on respiration.

12. There is a disposition in both functions to rise at the wonted hours of rising, as seen in the short fast.

13. On several occasions the rate of both functions suddenly halved or doubled itself, as in Case 35, 4th day, Case 33, 2d day, Case 34, 1st day; all at the same hour, viz., 2 p.m.

14. Upon the whole the rate of the functions was the least disturbed in the night and the working day, and then the effect of the respective influences was the most perfect, whilst the characteristic of the morning and evening was "change."

15. The ratio of respiration to pulsation was on the average not so low as 1 to 4 nor so high as 1 to 2·3; but it was much higher in women than in men; and in the case having the highest ratio it varied from as 1 to 1·4 to as 1 to 2·3, and in that having the lowest it varied from as 1 to 2·8 to as 1 to 5·8. It was the highest at night (from about 11 p.m. to 5 a.m.) and the lowest from 10 a.m. to 5 p.m. It was lowered directly after a meal and raised before a meal, and was the highest with sleep in the night.

The following are the chief variations from a state of health:

1. The rate of both functions is greater.

2. The night pulsation is scarcely beyond that of health; but the day pulsation is upwards of 20 pulsations greater, and is more than double of the healthy increase. Thus the chief variation in pulsation refers to the day.

3. The ranges of pulsation at each of the four periods of the day are much greater, but especially so in the "evening" and "morning."

4. The respirations in the night and also in the day are directly opposed; but both in health and disease they are increased generally after meals.

5. The effect of food upon pulsation is not widely different. It is a little less at breakfast and a little more at tea and supper, whilst it is equal at dinner. The effect upon respi-

ration was greater at dinner, less at breakfast, and equal at tea.

6. The ratio of the two functions is much higher, and is opposed in its hourly changes, since it is highest in the night and lowest in the day.

7. The effect of sleep is greater, and probably of sunlight also.

But in making the above statements we must not forget that the investigation for disease was made in June, with powerful sunlight, and that for health in November, with but little sunlight power.

Deductions.

The chief deductions from this investigation may be made with more truthfulness when I shall have ascertained the quantity of air inspired. Only a few will, therefore, be now presented.

1. As profuse perspirations occur in phthisis during sleep, and as, during both day and night sleep, the pulse is lowered, and that to a very great extent in the night, it is probable that the former much depends upon the latter. With this impression I have administered food with wine or cold tea in the night with great advantage in preventing the perspirations. As milk scarcely increases pulsation, it has not been of great service for this purpose. Further, as day sleep is accompanied by a rate of pulsation much above that attending upon night sleep, it is probable that the exhaustion which induces the perspiration would be somewhat prevented by cultivating day sleep and curtailing the hours of night sleep. As the pulse falls in the evening and rises in the early morning, under all circumstances, I believe that to retire to rest at 8 or 9 p.m. and to rise at 6 a.m. would save the system in phthisis; but with the early rising it is essential that there should be an early breakfast.

2. As the influence of sunlight is so powerful at Midsummer in increasing the rate of pulsation so much above

the condition of the pulse in darkness, it may be inferred that too free exposure to it would exhaust the system. On the other hand, the deficiency of sunlight in winter, and more particularly in close, dark streets, alleys, and rooms, is likely to maintain the state of low vitality which is so essential a part of the disease. Free exposure to sunlight in winter and restricted exposure in summer seem therefore to be indicated.

3. The extremely low rate of pulsation obtained and maintained for so many hours of the night, with, at the same time, the greatly increased rate of respiration during that period, indicate the necessity for the administration of nutritive food once or more frequently through the night. I believe this to be very important in preventing the exhaustion attending upon low pulsation, and also as permitting the meals taken during the day to be moderate in quantity, and thereby to prevent much of the unnatural and exhausting day elevation of the pulse. To this end it is probable that the administration of carbonaceous matter, as cod-liver oil, the last thing at night, immediately before the great increase in the rate of respiration from sleep, would be beneficial. But the night increase in respiration is not found in every case.

4. The ratio which the respiration bears to pulsation must be important in phthisis both in prognosis and in treatment. This, other conditions being similar, is influenced differently in different cases. Thus in one the ratio shall be lessened by increase of pulsation from excitement during the day, whilst in others excitement shall have but little influence, and in them the ratio shall be lessened by very profound sleep, which lessens pulsation and increases respiration. In order, therefore, to valuable deduction, it is needful to compare the day with the night, and not to trust to absolute numbers. However injurious rapidity of both functions may be, it cannot be but that rapid pulsation with slow respiration must be more so. Hence, when the ratio is so low as 1 to 7 or 1 to 8, it is open to inquiry if such a condition will not necessarily lead to impoverishment of the

system. Hence the aim would be to increase respiration disproportionately to pulsation. This is probably effected by remedies which give tone to the system; but it is now proved to be so by food, and especially by sleep, and hence the due cultivation of the horizontal posture with frequent food and day sleep are indicated. Early retiring to rest has also the same effect. I have noticed the low ratio referred to chiefly in phthisical persons of unusual stature, as in some of the regiments of Life Guards, and in the early stage. Women appear to obtain a sounder sleep than men, for in them the pulse is more quickly reduced and reduced to a lower point at night, whilst, at the same time, the rate of respiration is greater than in men. The cause of the night increase in the rate of respiration in phthisis cannot be ascertained until the quantity of air inspired is determined.

EXPLANATION OF THE DIAGRAMS ACCOMPANYING
THE PAPER ON THE HOURLY RATE OF PULSATION
AND RESPIRATION IN PHTHISIS.

Nos. 1 and 2 are constructed on one principle, and refer, the former to three men, and the latter to three women. They represent the rate of both functions, in the lying posture, at each of 144 consecutive hours, or six days and nights; the upper series of lines in each diagram being devoted to the pulsations, and the lower to the respiration in each case. The series of vertical double lines indicate the hour of midnight, whilst the intervening spaces are each divided into twenty-four columns, each indicating one of the twenty-four hours of the day. The thinner vertical lines show the hours of meals in each case on every day; and the horizontal lines, with the scale on either hand of the diagram, indicate the rate of both functions at every hour of the inquiry. The shaded parts include the hours of darkness in May and June, and the white parts the period of sunlight. The letter S shows that the patient was asleep at that hour, whilst $\frac{1}{2}$ S signifies half-sleep, or a state which did not appear to the observer to be true sleep.

No. 3 exhibits the same facts, on an average derived from 144 hours. The six outer figures are devoted to the six cases, and the central figure to the average of the whole combined. Each figure resembles a clock-dial with twenty-four radii or hours, the outer boundary line showing the number of pulsations, and the inner line the number of respirations at each hour. Each radius or hour is numbered, and the scale of pulsation and respiration is attached to the concentric circles. The shading indicates the hours of darkness, and the white part the hours of sunlight. The period of meals is also given on each figure.

No. 4 shows, in the upper part, the average hourly respirations, in the lying posture, in each of the cases, with the averages of all the cases of each sex, and the hours of darkness and of light; and in the lower part the average hourly temperature of each of the two wards occupied by the cases under inquiry.



1000

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CASES
OF
PARAPLEGIA
ASSOCIATED WITH
GONORRHOEA AND STRICTURE OF THE URETHRA.
BY
WILLIAM GULL, M.D.

Received Feb. 7th.—Read March 11th, 1856.

IN the year 1833 Mr. Stanley read before the Society some cases of paraplegia arising from primary disease of the urinary organs.

According to the views expressed by him, disease of the kidneys may produce a morbid impression upon the cord through sentient nerves, which being reflected outwards to the extremities, may occasion an impairment of both motion and sensation, and paraplegia result without organic lesion of the cord itself.

It would require a more minute examination of the cord than was made in the cases given in the paper referred to before the important negative assumed in their explanation could be regarded as established, since it is known that the structure of the cord may be extensively disorganized, where an experienced observer, without the aid of the microscope, may fail to discover the traces of disease. In proof of

this I may quote the following case, which was under the care of my friend and colleague, Mr. Hilton, who has kindly placed it at my disposal.

Paraplegia following gonorrhœa and syphilis. Inflammation of the substance of the cord. No traces of lesion discoverable without the microscope.

James L—, æt. 20, admitted into Guy's Hospital, March 14th, 1855; a gentleman's servant, unmarried. Always had good health until he contracted gonorrhœa and had a chancre eight months ago. He was under treatment for three months. After the chancre had healed, he again became infected, and ulceration followed at the seat of the old cicatrix. For this he was again under treatment until the beginning of the year 1855. At that time (January 18th), having occasion to go from home, he slept, as he thinks, in a damp bed, and three days afterwards began to have pains and weakness in the legs and about the neck and occiput.

On the 26th he had a rigor, and the weakness of the legs was rather suddenly increased, with loss of sensation above the ankles and formication in the feet. Incontinence of urine came on at the same time.

On the 28th he managed to get down stairs with the help of his mother and the use of a crutch; but at night he had lost all power in the legs, and was carried to bed. During the next fortnight the loss of sensation gradually extended upwards to a line corresponding to the distribution of the ninth dorsal nerve. The sphincters were paralysed. The susceptibility to the excito-motor movements continued to increase, and the cord at length became so irritable as to occasion the patient great distress; the least agitation or the slightest touch bringing on violent spasmodic contractions of the legs, though the irritation was quite unfelt. There was a painful sense of constriction across the chest. Bed-sores formed and rapidly extended.

There was no important change in his symptoms until April 18th. The urine was ammoniacal and continually



dribbled from him, excoriating the scrotum and inner parts of the thighs. The bed-sores sloughed. There were frequent involuntary spasms of both legs ; but especially of the left. At this date he began to have cough, headache, and more frequent rigors. Tongue became furred. Pulse accelerated.

He died rather suddenly May 16th (four months from the beginning of his symptoms), having during the last month become much exhausted from frequent rigors and hectic.

On a *sectio cadaveris*, the vertebral canal was healthy. On opening the dura mater the two layers of arachnoid were found united, as usual, on the posterior surface of the cord by delicate adhesions. There were some osseous plates on the visceral layer of the membrane. No traces of vascular injection or of inflammatory exudation. *The cord had the normal size and appearance, and neither to the touch nor on section presented any obvious softening.* With a lens of an inch focus the surface of the columns at and below the origin of the sixth nerve had a mottled appearance, some portions being opaque and yellowish ; and a more minute microscopical examination discovered extensive disorganization of the nervous structure, the focus of the morbid change being at the middle of the dorsal region and principally in the anterior columns. The fibrous structure was loose, and amongst it, and apparently resulting from its disorganization, were numerous oily granules, together with a great number of the characteristic mulberry masses (*granule-cells*). Sections of the cord at the lower part of the dorsal and in the cervical region gave the same results, but in a less degree.

This proves that we ought to look with great mistrust upon the evidence which the unassisted eye supplies in the examination of nervous structures, where but slight lesions produce such decided and striking symptoms.

The following cases seem to show that, instead of regarding the nerves as the channels through which the cord is

secondarily affected in disease of the urinary organs, we ought rather to look to the *veins* or *the blood itself* as the means by which the lesion is propagated, and, instead of attributing the paraplegia to functional depression of the nervous energies, to refer it to inflammatory changes.

In the following case this pathological relation certainly existed. For the particulars of it I am indebted to my friend, Dr. Habershon.

Paraplegia. Acute spinal arachnitis and softening of the cord following retention of urine from stricture.

William W—, æt. 29, a cabman, admitted into Guy's Hospital on Sunday morning, September 19th, 1847, for retention of urine and stricture, to which he had been subject for several years. After a warm bath, and with some difficulty, the smallest catheter was passed and the urine drawn off. On the following day he had again difficulty in emptying the bladder, and twenty leeches were applied to the perinæum. From this date until the 28th the stricture was dilated daily, and he was going on favorably, being a considerable part of the day up and about the ward, apparently in his usual health. On the 28th he complained of a fixed and constant pain near the angle of the tenth rib on the right side, for which a blister was applied, with relief. Three days after (October 1st) he was free from pain, but feverish. He dressed himself as usual and sat by the fire; but, on attempting to return to his bed in the afternoon, he suddenly found his legs weak and numb. Pulse 120. Tongue thickly furred. He was freely purged without benefit. On the 3d the loss of sensation and motion was complete in both legs, and sensation was imperfect on the surface of the abdomen as high as the umbilicus. He had no pain in the spine, nor any convulsive movements of the legs. The bladder was emptied morning and evening by the catheter. In the intervals it dribbled away, highly ammoniacal and purulent. Mr. Key, under whose care the patient had been admitted,



saw him on the 5th, and considered the paralysis to depend upon thickening of the posterior common ligament.

He gradually became more prostrate. A large slough formed over the sacrum. The evacuations passed involuntarily. He expired on the 27th, one month from the commencement of the spinal symptoms. There was no affection of the brain throughout.

Sectio cadaveris.—Head not examined. On removing the cord with its membranes from the canal a small quantity of pus was found lying on the outside of the sheath, opposite the bodies of the sixth, seventh, eighth, and ninth dorsal vertebrae, and one of the vertebral veins in the lumbar region was full of well-formed pus. The spinal fluid was densely coagulable. The arachnoid was thickened and presented traces of recent inflammatory exudation. The dorsal portion of the cord was very distinctly and generally softened. Lungs healthy. Pericardium contained a small quantity of fluid, with a patch of fibrin upon the ventricle. Liver healthy. Kidneys large and congested, with spots of ecchymosis. Secreting structure coarse and soft. Pelves dilated; their mucous membrane and that of the ureters and bladder injected and covered with purulent exudation. An old stricture existed at the commencement of the membranous portion of the urethra, and several false passages, one opening into an abscess behind the bladder, and two returning into the bladder. The vesical veins in the neighbourhood of the pelvic abscess were thickened and partially obstructed by recent lymph. No traces of peritonitis. Intestines healthy.

Here phlebitis was no doubt caused by the catheterism, but in the following cases gonorrhœa probably brought on paraplegia as one of its proper sequelæ, after the manner in which its other secondary affections, as swelling of the joints, are produced. Whether this be through a purulent infection of the blood, or through some more specific taint, is yet unknown.

Paraplegia, with softening of the cord, following gonorrhœa and chronic gleet.

Henry F—, æt. 21, a pale and delicate man, a shoemaker. Habits irregular. Has had gonorrhœa many times, and is subject to a permanent gleet, increased when he indulges in drink. His general health has been good; and he was, so far as he knows, quite well on Tuesday morning, March 1st, 1853. In the afternoon of that day he began to have pain between the shoulders, and a diarrhœa came on, to which he had been frequently subject. This continued during the night, with increased pain in the back and spasmodic tremblings in the legs. Towards morning the legs became weak and numb, and he was unable to void his urine. His friends, for his relief, applied hot fomentations to the feet, legs, and pubes, which produced extensive vesication. He was brought to Guy's Hospital, March 4th, and admitted under the care of Mr. Bransby Cooper, with the following symptoms: complete loss of motion below the sixth dorsal vertebra; the muscles of the seventh intercostal space do not act in respiration; sensation perfect above the line indicated, but on the abdomen pinching or pricking the skin gives no pain, and only the faintest sensation; in the legs there is complete anæsthesia; bladder distended, with dribbling of urine; great exhaustion; pulse 110, weak; respiration tranquil, 24; febrile heat; tongue injected; complains much of thirst. Spine quite straight. A slight degree of tenderness and some sense of stiffness between the shoulders. At the epigastrium and about the penis, thighs, and ankles the integuments are vesicated and the skin is in parts sloughing, from the hot fomentations which have been applied, and last night, in addition, several bullæ formed spontaneously on the left ankle and on the soles of the feet. No bed-sore. Fæces healthy; passed involuntarily. Urine drawn off by the catheter; ammoniacal, and containing mucus and pus, with traces of blood.

He died exhausted from irritative fever and sloughing,

March 15th, a fortnight from the beginning of the paraplegic symptoms.

Sectio cadaveris.—Several superficial sloughs over the legs and abdomen. Large, sloughing bed-sore over sacrum. Bullæ on the soles of the feet. Bones and ligaments of the spine healthy. The cord was generally softened as high as the middle of the dorsal region, at which point the nervous substance was broken up by the gentlest stream of water falling on it. The gray and white portions appeared to be equally affected. Amongst the softened nerve tissue granule-cells were abundant. There was no point of suppuration, nor any trace of old disease in the cord. The membranes were apparently healthy; liver healthy; kidneys of a dark colour, from venous congestion; the mucous membranes of the pelves slightly ecchymosed; bladder thickened, the lining membrane covered by recent diphtheritic exudation. Between the bladder and rectum there was an irregular abscess, with sloughing walls, communicating with the bladder by a large perforation of its coats. Near the bulb was a more recent abscess, filled with healthy pus. The lungs collapsed freely on opening the chest, and were free from disease. Heart healthy.

Paraplegia occurring with gonorrhœa. Recovery.

Alfred L—, æt. 28, a thin, pale young man, below the middle stature, employed in a ready-made-shoe warehouse. His habits are rather irregular, but yet he has had no illness until the present attack. On the 17th of June, 1855, he took a gonorrhœa, attended with the usual symptoms, until four days ago, July 1st, when he began to feel pain between the shoulders, which he attributed to cold, though he knew of no exposure. The following day the pain had increased, and extended to the loins, but was not such as to prevent his going on a Sunday excursion to Brighton. On reaching town in the evening, he walked to his home, the distance of a mile and a half, without feeling

any weakness in his legs. The next day (July 3d) he was at his work, but felt very unwell, and his legs weak; he had some difficulty in emptying the bladder. In the evening he applied a mustard poultice to the loins, and passed a quiet night. July 4th, he was unable to leave his bed, and sent for my friend, Mr. Roper, on account of retention of urine. During the day the legs became weaker, and in the evening he again required the use of the catheter, and his evacuations passed from him involuntarily. July 5th, he was admitted into Guy's Hospital, under my care. He could move the legs only very feebly; numbness along the outer part of the thighs, as far as the knees; involuntary muscular twitchings in both legs; abdomen flaccid, a sense of constriction around the lower part; inability to empty the bladder; constipation; no tenderness in the spine; no affection of the joints. Tongue moist, slightly furred; skin cool; pulse 76. Moderate gonorrhœal discharge.

By cupping, laxatives, and counter-irritation, the paralytic symptoms slowly disappeared, and on the 15th he was able to stand without help, though not to walk. A third blister was applied to the loins, and medicine continued, until he became impatient to return home. When he left the hospital he had but slight weakness in the legs. The gonorrhœal discharge had gradually ceased.

He continued to attend as an out-patient for a month, and was then apparently well.

I saw nothing more of him until July, 1856, when he again applied on account of a slight return of his spinal symptoms, of which he maintained he had no traces until the occurrence of the gonorrhœa.

By a return to his former treatment, he again improved, and is now well.

That the occurrence of paraplegia with gonorrhœa is due to an inflammatory affection of the cord is further made probable by one at least of Mr. Stanley's own cases, which I may perhaps be allowed to quote in this place:

"A man, æt. 30, was admitted three weeks previously on account of gonorrhœa, with phymosis, which was in progress towards cure; the inflammation in the urethra had subsided, but the discharge continued. Whilst in this state as far as the local disease was concerned, and without any particular derangement of the general health, he was suddenly seized with paraplegia, which extended as high as the umbilicus. In the limbs, the loss of motion was complete, and the loss of sensation nearly so: the functions of the brain were unaffected. On being questioned, he stated that he had been suffering for a day or two from pain in the loins. The pulse was 85, and rather full. He was cupped in the loins, and free action of the bowels obtained by purgatives, but with no benefit. The urine flowed involuntarily, and in considerable quantity. As, however, it was thought the bladder was distended, a catheter was introduced, and three pints evacuated. In sixteen hours from the attack of paraplegia, the man suddenly fell back in his bed and died. The spinal cord was first carefully examined. *There was found some turgescence of the vessels both in the membranes and substance of its lumbar portion*, and a few drachms of transparent fluid in the theca, 'but,' says Mr. Stanley, 'neither the turgescence of vessels nor effusion of fluid was sufficient to explain the paraplegia by pressure on the cord.' The liver was enlarged and indurated. The other abdominal viscera, with the exception of the kidneys, were sound, and with no unusual turgescence of the vessels. Both kidneys were of so dark a colour as to be almost black; they were remarkably flaccid, and, on sections being made of them, were found to be in every part gorged with blood. The mucous membrane lining the infundibula and pelves was dark-coloured, from the turgescence of the vessels. The coats of the ureters, and the mucous membrane lining the bladder, were also very much more loaded with vessels than is usual. In the bladder was about a pint of urine. Some fluid was found between the membranes of the brain and in its ventricles."

The objection made by Mr. Stanley that the turgescence

of the vessels and the effusion of fluid were insufficient to produce paralysis by pressure is no doubt valid, but it equally applies to a large proportion of the cases of paraplegia from inflammation of the structure of the cord. It is not often the amount of exudation which, by its mechanical action, determines the paralysis, but rather the coincident changes in the nervous tissue from defective nutrition and softening. The small amount of exudation, and the apparently slight changes of structure which accompany the inflammatory lesions of the cord, is one of the most remarkable points in their history; and here it is, as I wish to prove, that the microscope has so much aided our investigations. With the knowledge we have so obtained, the *injection of vessels in the structure of the cord* must be considered an important indication of organic lesion, and can leave us but in little doubt that the paralysis was the result of disease of the cord, rather than of any simply morbid impression made on it through incident nerves.

CASE OF A YOUNG WOMAN
IN WHOM
THE MAIN ARTERIES
OF BOTH
UPPER EXTREMITIES AND OF THE LEFT SIDE OF THE NECK
WERE THROUGHOUT
COMPLETELY OBLITERATED.

BY
WILLIAM S. SAVORY, F.R.C.S.,
TUTOR AND DEMONSTRATOR OF ANATOMY AT ST. BARTHOLOMEW'S HOSPITAL;
SURGEON TO THE ROYAL GENERAL DISPENSARY.

COMMUNICATED BY
EDWARD STANLEY, F.R.S.

Received Feb. 20th.—Read March 25th, 1856.

ANNA MARIA W—, æt. 22, was admitted into St. Bartholomew's Hospital, under the care of Dr. Burrows, on November 27th, 1854. She complains of general debility and obscure pains in various parts of the body, particularly of severe pain on the left side of the head, which from time to time becomes considerably aggravated. She often feels very giddy; the vision of the left eye is indistinct. There is a sense of constriction and uneasiness in the left region of the chest, with occasional palpitation. The slightest exertion

causes great dyspnœa; at the same time, the limbs of the right side become agitated and powerless.


She is a very delicate-looking young woman; countenance flushed; lips dusky; remains of herpes about nostrils; skin moderately warm, soft, and moist; tongue nearly clean and red; bowels constipated; urine natural; no appetite; catamenia scanty and irregular; she sleeps very well.

No pulse can be detected in any part of either arm. There is a feeble pulsation in right carotid; it is doubtful whether it can be felt in the left; both femoral arteries pulsate feebly; the heart beats 96 times in the minute. The temperature is not unnaturally low in any part of the body.

The formation and movements of the chest are healthy. There is no abnormal dulness in either the pulmonary or cardiac regions; heart's impulse increased; no murmur audible at its apex; there is a loud, harsh, systolic bruit over the top of the sternum, which gradually disappears as it is traced downwards along the aorta to the heart; there is a soft bruit in the course of the right common carotid.

At this time she gave a very obscure and imperfect account of her history. Much of the following information I have lately obtained from her mother, and other sources.

Until five or six years of age, she was a healthy child. Since that time she has been delicate, constantly requiring medical care. She has been a patient in various hospitals. During the last nine years, the catamenia have been frequently irregular. Her strength has rapidly declined, and for the last five years she has repeatedly experienced temporary loss of power of the left side. I can obtain no certain evidence of the precise period when the pulse first ceased at the wrist. Her mother, who is a very intelligent woman, says she is certain it has not been felt for the last five years, and the girl herself confirms this statement. In 1853, from May 18th to August 9th, she was a patient in Guy's Hospital, under the care of Dr. Barlow. Dr. Wilks has most kindly furnished me with the following information



concerning her condition at that time. She was admitted for chlorosis and deranged menstruation. She then stated that the catamenia had first appeared six years ago, and that they had not been regular since. At that time, no pulsation could be distinguished in either brachial artery, or in any of the branches below. She was aware that no pulse could be felt at the wrists, and stated that it had ceased to beat for some years.

Since that time she has suffered from pain in the right arm, which was supposed to be rheumatic. Eighteen months ago, she was seized with involuntary, and occasionally violent, shaking of the right arm. Shortly afterwards, the corresponding leg became similarly affected; both extremities of that side subsequently became very weak; then followed pain in the head. Three months ago, she became an out-patient at St. Bartholomew's Hospital. At that time no pulse could be distinguished in any of the vessels of the head, neck, or upper extremities; the femorals and the vessels of the lower extremities pulsated, but not strongly; a bruit could be detected in the right common carotid. A shrill bruit, almost amounting to a soft whistle, could be heard at the top of the sternum. Beyond this no morbid sound was detected. Lately, during her attendance, the left eye became weak, and its vision imperfect.

On November 27th she was admitted into the hospital as before mentioned.

She was ordered milk diet, with beef tea, wine, and quinine.

On the 29th and 30th, she complained of severe pain in the lumbar region. There is evidently a great tendency to hysteria; she is so weak that she sits up with great difficulty. The vision of both eyes is very weak and imperfect, but it is decidedly much better when she lies down; in this position she can see the length of the ward, but when sitting up she can scarcely see at all. The sclerotic coat of the left eye is congested.

December 1st.—A slight squamous eruption has appeared on the head, and she says her hair is falling off rapidly.

16th.—No particular change has occurred since the preceding report. During the last few days she has complained of severe pain in the left parietal region, more especially in a small spot to which the squamous eruption is limited; this pain is now increasing. She complains that her legs feel very cold; pulse at heart, 110; thirst intense.

She was ordered—

Hydrargyri Chloridi, gr. ij;
Opii, gr. ss; omni nocte.

Potassii Iodidi, gr. iij, e Decoct. Sarzæ, ter die.

Unguent. Hydrarg. Nitratis Capiti.

23d.—The pain over the left parietal eminence still continues to be very severe, and it extends down towards the ear; the sclerotica of the left eye, which, during the last few days, has been less vascular, is now inflamed, and there is a small ulcer at the lower border of the cornea.

She was ordered meat diet; the wine and mixture were continued. A solution of nitrate of silver was applied to the eye.

30th.—In many respects she feels better. The left eye is nearly well, but the pain in the left side of the head has rather increased, and now there is great tenderness in the region of the left parietal eminence. She complains also of pain at the top of the sternum, and is harassed by a troublesome cough, accompanied by very scanty mucous expectoration. The systolic murmur at the top of the sternum is becoming softer.

The same treatment was continued.

January 6th.—She has been more cheerful since the last report, and has tried to sit up, but was soon compelled to lie down again, feeling giddy and weak; vision still continues to be very imperfect, and there is great intolerance of light. The murmur is louder in the region of the arteria innominata, and perhaps a faint bruit is audible in the course of the left carotid. The murmur in the right carotid still continues.

13th.—She has been up for a short time since the last report, but complained of distressing vertigo ; she declares that she completely loses her sight when she attempts to stand. The pain and tenderness of the scalp still continue ; there is a slight, puffy swelling of the integuments. She is extremely languid and exhausted ; there is a dark areola around each eye ; she is gradually becoming weaker.

February 3d.—She has complained during the last few days of loss of power on the right side ; the sight of the left eye appears to be almost gone ; the catamenia have occurred this week. Ordered—

Essen. Sarzæ, ʒss, e Lactis cyatho, ter die.

The Iodide of Potassium to be discontinued.

10th.—The pain in the head continues unabated ; she has lately passed such bad nights that she has been ordered to take morphia in the evening, which has lessened the pain and given rest ; the skin over the left parietal eminence has yielded, and a scab has formed, attended with a slight sanious discharge. Ordered—

Olei Morrhuæ, ʒss, bis die.

17th.—Nothing worth recording has occurred since the last report. There is, perhaps, less pain and tenderness in the scalp ; the scab remains, from beneath which a brown, half-purulent discharge exudes ; the integuments around are inflamed ; there has been for the last few days slight ulceration about the septum nasi ; the catamenia are present. Poultices were ordered to the scalp.

21st.—The left side of the head is painful and extremely tender ; the discharge from the scalp has increased, and become more purulent ; the ulceration about the septum nasi has extended.

24th.—The pain in the head increases ; there is superficial ulceration over the left parietal eminence, which discharges pus ; the action of the bowels has been lately

irregular, and their contents unhealthy; she sleeps very badly.

The same treatment to be continued.

Fil. Hydr. Chlor. ē. Colocynth., alter. nocte.

March 3d.—Since the last report the pain in the head has been exceedingly severe; she usually leans forward in bed, supporting the head with both hands; there is a circular ulcer in the scalp about the size of a shilling, with a clean surface, discharging freely; the ulceration about the nose has improved. Lately, she has been attacked by severe shaking fits, which involve the whole body, and which are succeeded by impaired and disordered sensation.

6th.—The ulceration of the scalp has extended, and exposed the bone; it is less tender; the application of poultices gives relief.

13th.—Since the last report she has grown weaker, and suffered severely from pain in the region of the ulcer; the surface is covered with pale, flabby granulations, and the exposed bone at the bottom is stained of a brown colour; the discharge is about the same in character and quantity; the left eye is still inflamed, and vision is very imperfect, with intolerance of light. The shaking fits recur every day.

15th.—In the evening she was suddenly seized with loss of power and sensation on the right side. It remained for about an hour, and then passed away.

April 9th.—Nothing particular has occurred since the last date. She grows weaker, otherwise her condition is in most respects the same; her eyesight, particularly the left, has almost gone; temporary suspension of power and sensation still occurs, and it is preceded by confusion of thought and memory; the ulcer on the head extends, and looks unhealthy; more bone is exposed.

She was ordered a liberal supply of nourishment and wine.

June 4th.—Since the last report she has had more

nourishment, and porter, and has felt somewhat better; but the ulcer on the head has slowly extended and denuded more bone; the exposed portion is exfoliating.

10th.—A portion of dead bone, somewhat larger than a shilling, is slowly separating.

29th.—To-day a few drachms of blood escaped from the ulcer.

Shortly after this time the portion of dead bone came away. The ulceration had extended beneath it into the substance of the cerebral hemisphere. The whole surface had a pale, flabby aspect. Shortly after this a portion of bone, extending towards the right side across the vertex, became loose, but resisted gentle attempts at extraction.

She lingered on with but little variation in her symptoms for some months. Latterly she was removed to one of the surgical wards, under the care of Mr. Stanley. Towards the last the sensibility of the right side seemed occasionally to be unduly exalted. Her strength slowly passed away, and at last she sank on the 24th of December, 1855, having been a patient in the hospital for thirteen months.


NOTE.—For many of the foregoing details I am indebted to Dr. Trollope and Mr. Green, who were the clinical clerks at the time, and also to Mr. Dalley, Mr. Stanley's house-surgeon, who watched the case with much care.

December 26th.—*Post-mortem, forty hours after death.*—The body was only slightly emaciated. The limbs were thin; but they had a healthy and natural aspect.

On the left side of the head, over the parietal eminence, was the large circular ulcer, between one and two inches in diameter, which evidently had passed to some considerable extent into the cerebral substance. The surface presented the appearance of a slough. When the skull-cap was removed the inner table of the parietal bone was found to be extensively necrosed. The dead portion extended for some distance beyond the right margin of the aperture, and across the sagittal suture for half an inch or more into the right

parietal bone. The dead portion was separated by a well-defined margin from the living, and was loose, though sufficiently adherent at some points to prevent its ready removal. The dura mater was extensively destroyed. A circular portion, exceeding two inches in diameter, had disappeared. The edges of the aperture were sharp, and terminated abruptly. A vertical section through the corresponding portion of the left cerebral hemisphere showed the substance of the brain to be disorganized around the ulceration to the depth of an inch or more. The veins upon the surface of the hemispheres were turgid with blood. The sinuses contained not more than the usual amount of clot. The vessels of the cerebral substance were congested, and this increased vascularity was especially apparent in the neighbourhood of the ulcer. The ventricles contained very little fluid. No deviation from the healthy condition could be detected in the vessels at the base of the brain. The membranes, except at the part indicated, appeared healthy.

The heart, its muscular tissue, its investing and lining membranes, the cardiac and arterial valves, were healthy. The aorta, at its commencement, presented no appearance of disease. In the arch several small portions of the lining membrane were dull and opaque; and numerous, small, irregular, opaque, yellowish deposits existed in this part of the vessel. From this point downwards through the thoracic aorta the natural aspect of the internal coat of the vessel was destroyed. The lining membrane was uniformly dull and opaque. The coat itself separated from the middle tunic with morbid facility, and when carefully examined it was found to be decidedly thickened. It had lost its peculiar brittleness, and was tougher than natural. There was wanting that peculiar disposition to curl, which belongs to its healthy condition. No sensible change could be detected in the two outer coats, and the areolar tissue around the artery appeared unaltered. The morbid condition of the vessel gradually subsided as it passed through the abdomen, until at its bifurcation there was very little perceptible thickening of the inner coat. From this point downwards



into the femorals, although the arteries could not be pronounced absolutely healthy, yet there was less certain and obvious evidence of disease.

But in the *arteria innominata* the changes just described, and others too, were most strikingly shown. The whole vessel was very much thickened, and it required considerable pressure to approximate its walls. It was quite empty. The lining membrane was pale and smooth, but dull and opaque. The internal coat separated with the slightest force from the middle, and was almost as thick as the other two coats together. The middle and external coats were denser than natural, and slightly thickened. Owing to this morbid state of its tunics, a section of the artery presented a remarkable appearance. The distinction between the coats was strikingly obvious. When the internal coat was stripped off from the middle, in a great portion of the vessel irregular patches of an opaque yellowish deposit, in considerable quantity, were found between the two. The greater part of this deposit came off upon the outer surface of the inner coat, but some remained upon the inner surface of the middle. The deposit, to microscopical examination, presented all the characters of consolidated lymph, intimately blended with the arterial tissues. It was probably in process of degeneration, for here and there many small globules of an oily nature could be detected.

This morbid change extended into the right carotid, and the commencement of the subclavian artery. In the whole of the right common carotid the same change, although to a less extent, was apparent. The vessel was contracted and much thickened. There was no clot in its interior. The inner surface was not reddened. There was scarcely any perceptible change in the internal and external carotids. They were perhaps somewhat thickened and contracted.

About an inch or less from their origin the right subclavian, the left carotid, and the left subclavian arteries became suddenly contracted to one fourth or one fifth of their natural size. This change extended throughout these vessels, through the axillary, brachial, radial, and ulnar

arteries on both sides, and the left external carotid artery. The contracted canal in their interior was completely blocked up and obliterated by a fibrous cord, which extended with scarcely any interruption throughout their entire length. The left internal carotid artery was pervious; and, beyond being somewhat contracted and thickened, was not perceptibly changed.

Thus all the main arteries of both upper extremities and of the left side of the neck were reduced to solid cords, and presented the exact condition of vessels through which the flow of blood had been for some time mechanically arrested. The brachial artery was smaller than the median nerve. The areolar tissue around was not unnaturally dense: it appeared healthy. In a transverse section of one of the obliterated vessels the distinction between its contracted coats and the cord filling up the canal was obvious. The opaque yellowish-white coat contrasted strongly with the greyish cord within. A longitudinal section of the vessel displayed the connection between the coats and the cord. In many parts the adhesion was firm and resisting. In some parts it seemed impossible to separate the cord from the inner coat, which, upon the application of much force, came away with it, while in other parts no adhesion whatever was apparent; when the cord was removed the internal coat remained undisturbed. It was generally slightly wrinkled. There was no actual deposit upon its surface; but here and there it was perhaps roughened. The connection between the cord and the coats was firmest and most universal in the brachial arteries, and least so in the left carotid. Almost throughout, the inner coat of this vessel presented numerous longitudinal wrinkles.

The cord which filled up the canal commenced abruptly in each vessel by a blunt extremity, around which the coats became suddenly contracted.

When examined, it presented all the characters of simple delicate fibrous tissue, such as would result from lymph or fibrine.

Generally, at the origin of a branch, the cord was more



or less deficient, and the canal pervious for a few lines. In these situations what substance there was in the interior was confined to the opposite side of the vessel to that whence the branch arose.

The secondary branches of the subclavian, axillary, and brachial arteries, beyond being thickened and contracted for some distance from their origin, presented, so far as they were examined, no unusual appearance.

No morbid change could be detected in any of the veins. They appeared to be in all respects healthy and natural. The pulmonary vessels, the lungs, liver, spleen, kidneys, and the viscera generally, were remarkably free from disease. The spinal cord was, perhaps, somewhat more vascular than usual; otherwise there was no sign of disease. The muscles and the other tissues gave no indication of a deficient supply of blood. The upper extremities were as well nourished as any other portion of the body. The examination of the collateral channels, in those parts whose main arteries were occluded, was necessarily only imperfectly conducted; but those vessels which were traced were not visibly enlarged.

After the simple narration of this case, I think no further explanation of the reasons which have induced me to bring it before this society will be required. It is certainly one of the most remarkable on record, and is, I believe, in some respects, unique. The condition in which the arteries were found after death—the morbid changes which they had undergone—will generally, I imagine, be regarded as the result of inflammation of their walls. Under what circumstances this inflammation occurred—the nature of its symptoms, and the phenomena of its progress—concerning these points the history of the case is, unfortunately, somewhat obscure. But from all that could be ascertained, it appears to be tolerably certain that the disease, in no portion of its history, was ever accompanied by any well-marked or characteristic symptoms. The most careful cross-examination failed to elicit any account of such active and violent symp-

toms as are said to distinguish acute and general arteritis.¹ The condition of the arteries which was disclosed after death, had most probably been very gradually effected. The morbid changes in their progress must have occupied a considerable period; for there is satisfactory evidence, that during the last five years no pulse could be detected at the wrists. It is certain that in 1853, more than two years and a-half ago, when a patient in Guy's Hospital, no pulse could be detected in any of the arteries of either arm;² whereas, at the time of her death, in some vessels the morbid changes had not far advanced. The condition of the brachial arteries contrasted strongly in this respect with the two carotids.

The symptoms observed during the time she was actually under observation in St. Bartholomew's Hospital, may fairly be regarded rather as the result of the changes which the arteries had undergone, than as due to the progress of the disease itself. And these symptoms, when compared with the condition of the arteries which was disclosed after death, are most interesting and instructive.

They may almost all be referred to a deficient supply of blood to those parts of the body to which the arteries, which had become obliterated, were distributed.

The obliteration of the left carotid offers a satisfactory explanation of the destructive ulceration on the corresponding

¹ See 'F. Tiedemann von der Verengung und Schliessung der Pulsadern in Krankheiten,' 1843. Also the case described by Dr. Thompson in the appendix of Mr. Hodgson's work on the 'Diseases of Arteries and Veins,' 1815.

² Dr. Wilks was kind enough to point out to me the following notice of the case which he has published in 'Guy's Hospital Reports' for 1853:

"A girl, æt. 20, with chlorosis, had no artery to be felt in either arm below the subclavian. No brachial or radial could be felt. Whether this occurred from disease or was congenital could not be satisfactorily determined."

It is clear, from the last remark, that no history had at that time been elicited of any illness characterised by such symptoms as acute and general arteritis is usually supposed to exhibit.

The report above is from the pen of Dr. Wilks; therefore its accuracy will not be questioned.

side of the head, involving successively integuments, bone, and brain. It explains the congestion and inflammation of the left eye, and the ulceration of its cornea; and the destruction of parts about the septum nasi. No structures are so immediately dependent, for their healthy condition, upon a due and proper supply of blood, as the nervous centres. No organ is so readily affected by disorder of its circulation as the brain. A moment's reflection upon the effect which obliteration of a carotid must have upon the circulation in the corresponding cerebral hemisphere, will suffice to account for the vague and anomalous affections which distressed the poor girl while living; for the vertigo and impaired vision, especially of the left eye, which increased as she approached the erect position; and for the impaired motion and disordered sensation of the right side.

Perhaps, at first sight, it may appear remarkable that the upper extremities exhibited so little the effects of a deficient supply of blood. But besides being placed under more favorable circumstances in this respect, it is to be borne in mind that they were affected at an earlier period of the patient's history, while the bodily powers were much more vigorous, and more capable of sustaining, for a time, a diminished supply of blood to a part, until the circulation was restored by means of the collateral channels.

A similar explanation may be offered of a fact mentioned in the relation of the case. No diminution of temperature was observed in the upper extremities. If the obliteration of the arteries had been a gradual change, it may never have been perceptible. But, under any circumstances, the temperature of the part would have been subsequently restored with the collateral circulation.

This case certainly does not confirm the opinions which Rokitsansky has advanced on the pathology of arteritis. It is difficult to reconcile the changes which were observed with the account of the disease which he gives in his '*Pathologische Anatomie*.'

In this case, in those arteries which exhibited most clearly the alterations produced by disease in their walls,

the inner coat had decidedly undergone the most extensive change, and the external coat was the least affected. This was especially evident in the *arteria innominata*, in which the internal coat was the most thickened and otherwise altered. Moreover, in this vessel there were deposits of lymph in considerable quantity between the inner and middle coats. It is indeed conceivable that these changes were secondary to, and produced by, inflammation of the cellular sheath of the vessel which Rokitansky says "is alone capable of inflammation;" but at the best, it would be but a very unsatisfactory explanation of the changes which were discovered. To admit this, we must assume that in that portion of the arterial wall, to which inflammation, when present, was limited, the slightest traces of it remained; while, on the other hand, on the remote side of the middle coat—which, says Rokitansky, "in the larger arteries, as, for instance, in the trunk of the aorta, exhibits so great a thickness, together with such density of texture, that we are unable to comprehend how it can be permeated by an exudation, unless by the agency of an acute process"—the changes produced by inflammation were most remarkably displayed.

At all events, we are not driven to admit that the exudation which was found, had permeated the entire substance of the middle tunic for the reasons assigned by Rokitansky. He declares that the middle coat is incapable of inflammation, because it possesses no vessels. He says "the absence of vessels in the (yellow) circular fibrous coat, and more especially in the inner coat of the vessels, forbids our assuming the possibility of inflammation in these layers."

Now, it is well known that some excellent anatomists have traced capillaries into the circular fibrous coat, and even through it; so, as Virchow¹ observes, the question does not turn upon the permeability of the middle coat.

There can be no doubt concerning the origin of the fibrous

¹ 'Archiv für Pathologische Anatomie und Physiologie und für Klinische Medicin,' Erster Band, 1847, p. 286.

d which filled up the interior of the contracted vessels. was evidently the remains of the blood which had coagulated in the canal,¹ and which had undergone changes similar those that occur in the clot, which forms in an artery beyond the point where a ligature has been applied.

That this cord was formed solely from coagulated blood, which had undergone the usual changes, and that no portion of it was due to any exudation from the arterial walls, seems proved by the following facts :

Its adhesion to the internal surface of the vessel was only partial and imperfect, and such as it was, might be readily explained by the morbid condition of the arterial tunics. It was almost totally absent about the origin of secondary branches, and where not occupying the entire canal, it existed on one side of the vessel only.

¹ See 'Rokitansky,' op. cit.



ANALYSIS OF CASES
OF
AMPUTATION OF THE LIMBS
IN THE
RADCLIFFE INFIRMARY, OXFORD.

BY
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ONE OF THE SURGEONS TO THE INFIRMARY.

Received March 25th.—Read April 8th, 1866.

For some years past, beginning with 1838, the capital operations in the Radcliffe Infirmary, at Oxford, have been registered from notes taken at the time of the operation. The Admission Books before 1838 show (as I believe) all the cases in which primary amputation was performed upon In-Patients for accidents as far back as 1810: and these cases are included in the tables in this paper. The secondary operations are not noted in the Admission books; nor (except in very few cases) are those in which amputation was performed for disease.

In arranging cases of amputation for purposes of comparison, two large classes are easily distinguished, — Amputation for Disease, and Amputation for Injury; and many subdivisions may be made in each class. The different

proportion of mortality, according as the operation is in the upper or lower extremity, and whether near the trunk, or at a distance from it, is well recognised: and one former observer at least (Mr. James, of Exeter,) has pointed out that the mortality in disease varies according to the nature of the disease, as much as in accidents, it varies according to the nature of the injury, and the period at which amputation is performed.

AMPUTATION FOR DISEASE.

Diseases of Joints.

The Register of operations contains notes of the cases of 96 patients (59 male and 37 female) on whom amputation was performed for Disease of the Joints, — 57 in the Thigh, 21 in the Leg, 7 in the Upper Arm, and 11 in the Forearm.

In some few of the cases here classed as 'Diseases of Joints,' it must be to some extent doubtful whether the disease began in the proper structure of the joint, or in the neighbouring bone (134, 159), especially in cases of the ankle (37, 82, 112, 118, 239), and wrist (69, 254). At the late stage in which patients apply at a Hospital, it is often difficult to tell clearly; and in cases of doubt, the difficulty is seldom altogether removed by dissection of the limb.

Of 57 cases in which amputation was done in the Thigh for disease of the knee, 6 died from the immediate effects of the operation: —

A married woman, 43 years of age, in a *very* low state before amputation, died the 5th day, exhausted. (150.)

A girl of 15 died the 18th day, exhausted. (151.)

A young woman of 19 died the 15th day, exhausted, apparently from the effects of secondary hemorrhage on the 7th day. (159.)

A young man of 20 died the 24th day. Much blood was lost at the operation; and about two hours afterwards hemorrhage took place, for which the stump was opened.

He seemed to sink from the loss of blood, from which he never thoroughly rallied. An abscess was found deep among the muscles of the thigh, but no pus in the veins, nor any appearance of inflammation. The viscera of the thorax and abdomen were healthy. (183.)

A sawyer, aged 38, died the 15th day. Much blood was lost at the operation, from which he never fairly recovered. The lungs were free from tubercles; there was slight inflammation of the left pleura; general inflammation of the pericardium, which contained several ounces of turbid fluid, with flakes of lymph. The stump was foul and unhealthy, without union (except at some points of the integument), with pus in the saphena, femoral and external iliac veins: the internal and common iliac, and ascending cava seemed healthy. (194.)

A boy, 9 years old, died the 8th day. He was very low before the operation, suffering from profuse suppuration from several openings about the knee, and lower part of the thigh. He gradually sank from the time of the operation. (210.)

— and 5 others did not recover their health, so as to admit of their being discharged on the recommendation of the operating Surgeon:—

A man, 53 years of age, died in the 11th week, with “long-continued diarrhœa.” (7.)

A man, about 22, died in the 6th week, with phthisis; the stump was nearly healed. (59.)

A lad of 18 died in the 7th month, with diarrhœa. Ulceration of the ileum was found, with peritonitis; tubercles were thickly deposited throughout the lungs. (160.)

A man, aged 38, died the 38th day with phthisis. The stump healed quickly. Disease in the lungs, which had been quiet before the operation, was rapidly developed; and at his own request he was taken to his home in the country the day before he died. (230.)

A glover, aged 25, under my care, died in the 11th week, with large sloughs on the sacrum, and with phthisis,

— probably miliary tubercles without cavities. At the time of his admission, I proposed amputation as the only means of relief; but he did not consent till after seven weeks, by which time he had become much lowered by the constant pain of the disease, and by the discharge from sloughs which formed over the sacrum. The stump healed slowly. At his own request, he was taken to his home in the country, and died in two days. (250.)

NOTE. — In this case, and in three others (105, 208, 248), the articular cartilage had disappeared altogether from the outer condyle of the femur, while on the inner it was only partially absorbed or thinned. In two other cases in which it had disappeared from the outer condyle, it was sound on the inner in one case (224), and probably, also, in the other, as its appearance is not noted. (16.)

Of 21 cases in which amputation was done in the Leg, for disease of the ankle, or of the joints of the tarsus, all recovered from the operation.

One, however, died in the House: —

A boy of 15 died in the 16th week, with phthisis. The stump had healed some weeks. (70.)

All the patients who underwent amputation in the Upper extremity recovered from the operation, and left the Infirmary "cured."

Of those who recovered from the operation, and were discharged from the Infirmary, 20 never regained their former health, — 9 dying within 6 months of their discharge, 6 within 12 months, 3 within 18 months; 1 lingering rather more than 2 years, and 1, in whom the stump did not heal, lived about 4 years.

Of those who, as far as is known, did recover their health, 16 have since died; —

3 from causes not connected with the original disease; — one from accidental violence (a fall down a staircase), 5½ years after the operation (57); — one, after several

attacks of general dropsy, died comatose, with granular kidneys, 6 years after the operation (104); — and one, who was much exhausted by a succession of abscesses, was attacked, during the late epidemic of smallpox, with the disease in a severe form, and died, between 8 and 9 years after the operation (105).

One died with dropsy and lumbar abscess, between 6 and 7 years after the operation (22); — and 12 died with symptoms of pulmonary phthisis (one of them having also diabetes); 1 lived about 15 months, 4 lived about 2 years or a little longer, 2 lived between 3 and 4 years, one lived about 7 years, one about 8 years, 2 lived nearly 10 years, and one nearly 13 years.

The other patients, with the exception of two (58, 63), of whom nothing is known, are believed to be all now in good health. Among the number are the first two patients in the Register, — at a distance of 18 years from the operation.

The history of the patients after leaving the Infirmary has been taken sometimes from personal knowledge and enquiry; in some cases from the reports of friends or relations; and in many instances from the neighbouring Clergy and Medical practitioners, whose friendly help has supplied many points of information.

From a careful examination of the notes in the Register, it does not seem that the deaths after amputation, from causes connected with the operation, or the subsequent death from phthisis, at an early or late period after removal of the limb, is dependent on the length of time the disease had lasted before operation, or the extent to which the joint was disorganized by disease.

In the course of a long disease, varied with occasional periods of relief, the early history, and the symptoms attending the first appearance are often forgotten, and can hardly

be recalled to memory with sufficient exactness for correct medical registration. The note in the Register is in almost all cases alike, — that the disease began "without known cause," and had existed some months (or years) before the patient came to the Infirmary. In later cases greater pains have been taken to note the history of the first appearance. In 5 cases the disease has been attributed by the patient to local injury (125, 129, 136, 167, 231). In one case disease had been quiet, without hindering the patient from his work, for nearly 20 years, till slight injury 6 months before his admission (84). In 4 cases it is remembered that the pain came on suddenly (113, 114, 248, 249).

Taking the history of the disease from these notes, with as near an approach to accuracy as can now be made, the cases may be arranged in classes: —

Those in which the disease had existed less than 12 months at the time of operation, of which there are 42 cases, — 30 men and 12 women.

Those in which it was of longer standing, yet less than 2 years, of which there are 20 cases, — 13 men and 7 women.

And those in which it had lasted beyond that time, 34 cases, — 16 men and 18 women.

Thus it seems that in cases submitted to amputation in early stages of disease, the proportion of men is greater than that of women: in later stages the proportion of women is greatest.

Although it is not stated in every case whether it was the Right or Left limb removed, yet it is clear that the Right was the subject of operation more frequently than the Left.

Of the Right Lower extremity there were 38 cases, — 29 of the thigh and 9 of the leg. Of the Left there were 32, — 24 of the thigh, and 8 of the leg.

Two other cases of the Right Lower extremity might be added, in one of which the thigh was removed (207), and

in the other the leg (193), for deformity, the result of old disease of the knee and ankle.¹

In the smaller number of cases in the Upper extremity, the proportion does not differ much. Above the elbow there were 2 of the Right, and 4 of the Left. Of the forearm there were 7 of the Right, and 4 of the Left.

One class of cases requires separate notice, — those in which amputation was done in Boys and Girls under puberty, or about that age.

Of these there are 15. Three of them died in the House (70, 151, 210). One died 5 weeks after being discharged (109), one in 6 months (15), one in about 9 months (89), and an other probably as soon (58). One lived about 10 years (27). One was killed accidentally about 5½ years after amputation (57).

The remaining 6 are believed to be all living, having out-lived the operation for periods of 13 years, 12 years, 10 years, 9 years, 4 years, and 15 months.

Various Diseases.

Of other diseases of various kinds, some local and some of constitutional origin, there are 28 cases. In 13 cases, amputation was done in the Thigh, in 14 in the Leg, and in one at the Wrist-joint.

Malignant diseases, 5 cases.

Necrosis, 5.

Caries, 4.

Other diseases of bone, 2.

Diffused abscesses, with destruction of bone, 1.

Gangrene, 2.

Elephantiasis, 1.

Old ulcerations, 3.

Useless or inconvenient limbs, 5.

All these patients recovered from the operation, except in 2 of the cases of Malignant disease.

¹ In 9 cases, under my care in the Infirmary, of contraction of the knee from former disease of the joint, 7 were of the Right and 2 of the Left.

Malignant Disease.

The cases in which amputation was done for Malignant disease were, cancerous ulceration in 3 cases, fungus hematodes in one case, and cerebriform cancer in one.

3 were in the Thigh and 2 in the Leg.

One of each died in the House :

A man, aged 38, died in the 5th week after amputation of the thigh for cancer of the bones of the leg of long standing. Two or three days before death the pulse became rapid and the breathing impaired ; but it was not till within an hour of his death that any immediate change was anticipated. Examination of the body did not disclose anything beyond slight enlargement of the heart, and some minute tubercles in the lungs, — “ nothing in short to account for death.” (65.)

A widow, 63 years of age, under my care, died the 13th day after amputation of the leg, for cerebriform cancer of the tibia, which had existed about 8 months.¹ Early in the morning of the 10th day she was observed to be faint and pulseless, bathed in a cold sweat. From this time she gradually sank. Much fat was deposited about the heart, and the muscular substance was softened ; the mitral and tricuspid valves were much thickened, with deposit on their edges. The liver was soft and fatty ; the kidneys in an early stage of granular degeneration. No appearance of malignant deposit was detected anywhere, nor any enlargement of the inguinal or lumbar glands. (232.)

In one case disease reappeared, and the man died in about a year. (60.)

The other two are now living ; — one 13 years after amputation of the leg, for what is supposed to have been fungus hematodes of the bones of the foot (71), and one after amputation of the thigh, performed 18 months ago, for cancerous ulceration spreading into the tibia. (233.)

¹ Museum, Ch. Ch.

Necrosis.

Of 5 patients who underwent amputation of the Thigh for necrosis of the bones of the leg, 4 are now in good health; and one, though living, is crippled by deformity of the spine (the result of *mollities ossium*), which has come on since the operation.¹ (117.)

Caries.

Four patients (all of them women) underwent amputation for caries of the tibia, — 2 in the Thigh and 2 in the Leg.

In one, disease reappeared in the other leg, and in the sternum, and she died in about 2 years. (39.) One continued well for 2 years, when she was attacked with small-pox, and died in 3 days. (79.) One continued well for about 7 years, when she was attacked with uterine disease, and died exhausted by hemorrhage, about 8 years after the amputation. (94.)

In the remaining case the patient is in good health. The operation was done 16 months ago. (235.)

A man, who underwent amputation of the Leg for enlargement of the bone following injury, died 13 years afterwards, from the effects of some internal disease of the abdomen. (64.)

An intemperate man, 48 years of age, underwent amputation of the Leg, for disease resembling Elephantiasis, which had been 14 years in progress.² There was not any reproduction of the disease at the time of his death, 3 years afterwards, during an attack of typhus fever. (180.)

In the remaining cases the patients are all now living.

¹ Case mentioned, Stanley, 'Diseases of the Bones,' chap. vii, p. 113.

² Museum, Ch. Ch.

AMPUTATION FOR INJURY.

Primary Operations.

The Register contains notes of 35 cases of Primary Amputation, — that is, cases in which amputation was done soon after the patient's admission, without trying to save the injured limb. The Admission Books show 15 other cases; being, as far as I can learn, all the primary operations performed on In-Patients between 1810 and 1838, — the year in which the Register begins. After forming an opinion that the limb could not be saved, the period chosen by the operating Surgeon has been as early as could be conveniently done; in cases of collapse, not till after the patient had rallied. In one case this did not take place for 16 hours. (126.)

Of these cases, 6 were in the Thigh, 13 in the Leg, 16 in the Upper arm, and 15 in the Forearm.

Of the 6 cases in which amputation was done in the Thigh, 5 were fatal, — all of them cases of very severe injury, attended with circumstances little favorable to recovery: —

A man of 47, died the 22d day. He had nearly severed his leg from the thigh by falling with his knee bent on a scythe; he had lost much blood before admission, and gradually sank from the time of the operation. (87.)

A lad of 19, died in 24 hours. He had suffered a compound fracture from a train going over the middle of the thigh on a railway. Very little blood was lost. (140.)

A man of 70, died the 6th day. He had fallen with his knee bent on a scythe, which passed through the joint and separated the posterior two thirds of the condyles, with part of the shaft, from the rest of the bone.¹ The popliteal vessels were not wounded, and there had been very little hemorrhage. The operation was little more than com-

¹ Museum, Ch. Ch.

pleting the separation of the limb. He went on favorably till the 4th day, after which he sank rapidly. (171.)

A farmer's boy, aged 14, under my care, died the 22d day. He had suffered a compound fracture of the femur, with laceration of the soft parts, laying open the knee-joint, and also a comminuted fracture of the tibia and fibula, from falling into a thrashing machine. He lost much blood before and during the operation. On the 10th day he complained of a little shivering and sickness; from which time he gradually sank. The end of the bone had perished for about a third of an inch; there was a small collection of matter in the course of the femoral vessels, and an other among the adductor muscles; the femoral and iliac veins seemed healthy. Both sides of the chest contained yellow turbid fluid with flakes of lymph, and the pleura was covered with a coat of recent lymph. Deposits of pus of various sizes were observed in the base of both lungs. The heart and pericardium were healthy, as were also the viscera of the abdomen. (197.)

A laborer on a railway, aged 28, died the 23d day. He had suffered a compound comminuted fracture, on the railway. Much blood was lost before admission. On the 14th day, when the stump had nearly healed, he complained of headache. The next day he had a rigor, the pulse became quick, and the breathing hurried; and he complained much of pain at the epigastrium. The countenance became sallow and anxious; the stump opened, and the bone protruded; and he gradually sank. A small collection of matter was found among the adductor muscles. The femoral vein seemed healthy as far as its junction with the profunda; the profunda and its larger branches, and the external iliac, contained pus, with patches of lymph adhering to the lining membrane. The pleura on both sides was obliterated by old adhesions. Purulent deposits of small size were found, surrounded by masses of red hepatization, in the base of both lungs. The pericardium contained about 4 ounces of serum. (199.)

In the case in which the patient recovered, the injuries were confined to the leg; the knee-joint and the thigh were not injured.

All the patients who underwent amputation of the Leg recovered.

Of the 16 cases in the Upper arm 3 were fatal: —

A man, 30 years of age, died the 21st day. The injury was received from an explosion by gunpowder in a stone quarry. His death is attributed to "inflammation of the chest." (1827.)

A papermaker, aged 38, died the 4th day, — having gradually sank. His arm had been caught in the machinery of a mill; the wrist and elbow joints were both laid open, and the laceration extended into the axilla. (164.)

A young man, aged 20, under my care, died the 21st day. He was a passenger one winter's night in a train which came in collision with a train of coal trucks, meeting it on the same line of rails. The broken woodwork of the carriages caught fire: when this was quenched, he was found under one of the carriages with the hand and wrist much burned, the upper arm crushed, and the bone cut through about half an inch above the insertion of the deltoid muscle. The wound had nearly healed, when, on the evening of the 15th day, he had a shivering fit, and a more severe one the next day, followed by a quick and weak pulse, and hurried breathing. The wound opened, and the bone protruded. The skin became yellow; and he sank with symptoms of purulent infection. Permission to examine the body was refused. (198.)

Of the 15 cases in the Forearm only one was fatal: —

A lad of 19, died the 30th day. The right hand had been destroyed by machinery, and amputation was performed just above the wrist. Hemorrhage took place the next day; and the stump was opened. On the 8th day free hemorrhage again took place; the stump was opened again, and a large vessel tied. On the 16th, he complained of sickness, and pain at the epigastrium. Symptoms of effusion into the

chest came on; he got weaker, and gradually sank. The ends of the radius and ulna had perished, and projected from the open face of the stump. The large veins of the forearm did not show any unhealthy appearance. The right lung was covered with lymph, and compressed against the vertebræ by a large collection of purulent matter, mixed with flakes of lymph. In the base of the compressed lung were several small purulent deposits, surrounded by circumscribed masses of grey hepatization. The left side of the chest was healthy, and also the heart, pericardium, and abdominal viscera. (200.)

To this list of Primary Operations, 2 late cases might be added, where the patients, in losing their limbs, underwent all the dangers — though not the actual operation, — of a surgical amputation, — 1 in the Leg, and 1 in the Upper arm.

A laborer on a railway, aged 24, was admitted 27th December, 1850, under the care of Mr. Hitchings, with destruction of the right foot, and compound comminuted fracture of the tibia and fibula, about the middle of the leg, laceration of the integument over the knee, laying bare the patella, and two lacerated wounds of the scalp. Early in the morning, before daylight, he was found drunk lying on the rails. During the treatment the end of the tibia separated, and a piece of the patella was discharged. He left the Infirmary on the 23d of April, with a good stump.

A lad, aged 16, was admitted 22d July, 1852, under the care of Mr. Hansard; his right arm having been cut off a little above the elbow by a chaff-cutting machine. The projecting end of the bone was sawed off, the vessels tied, and the stump dressed. The lad was discharged on the 25th of August, with a good stump.

Secondary Operations.

There have only been 6 cases of Secondary Amputation; — 2 in the Thigh, 2 in the Leg, 1 at the Shoulder-joint,

and 1 in the Forearm. Three of the patients were not brought to the Infirmary till the period which would have been preferred for amputation was passed. All recovered, except a woman in whom the arm was removed at the shoulder-joint.

Of the 2 cases in which amputation was done in the Thigh, one was for mortification after compound comminuted fracture of the leg, and the other for gangrene, after simple fracture of the thigh-bone, with rupture of the popliteal artery and vein.

The patient in whom the arm was removed at the shoulder-joint, was a married woman, 45 years of age, under my care. She had scalded, or burnt, the arm and shoulder, 3 days before admission, during some "fit," or in an attack of mania. The operation was performed on the 7th day after admission. She refused all food after the operation, and became so unmanageable that the wound could not be protected from her violence. She died in 28 hours, — apparently sinking from maniacal exhaustion.¹ (234.)

This is the only case since the Radcliffe Infirmary was opened in which the arm has been removed at the shoulder-joint. In 4 late cases the operation might have been done, if it had been thought desirable so far to disfigure the patient. (137, 164, 196, 198.) In one of them there was a fracture through the neck of the bone, beside the injuries for which amputation was performed. (196.)

One other case might be added to these Secondary Operations, in which the patient underwent the danger, though not the actual surgical operation.

A boy, aged 13, was admitted 14th February, 1856, under the care of Mr. Symonds, with his left foot and ankle crushed in a thrashing machine 3 days before admission.

¹ The case is fully reported in the Asylum Journal, July, 1855.

The foot, which was gangrenous, was afterwards separated. The wound healed quickly, and the boy was discharged on the 9th of April, with a good stump.

There is not anything among the records of the Infirmary which will show how often a life has been lost in trying to save an injured limb. To supply this want, since my connection with the Infirmary, I have noted every case in which a patient has been admitted with any injury of the extremities which ended fatally. Through the kindness of my colleagues, who have afforded me every facility in this investigation, I have obtained notes of the cases under their treatment as well as my own. During this time, 17 patients, admitted on account of injuries in the limbs, more or less severe, not complicated with any injury of the head or trunk, and wholly within reach of amputation, have died from the effects of the injuries received.

In 3 of the cases the patient's life would perhaps have been saved by primary amputation of the Leg. In one the operation was advised, and the patient refused to submit.

A guard on a railway, aged 40, was admitted 22d November, 1851, under the care of Mr. Cleoburey, with the left leg crushed by a train going over the middle of the limb. Immediate amputation was advised, which was refused by the patient. Gangrene followed, and the man died on the 28th. This is the only case within my knowledge in which immediate amputation has been advised, and refused by the patient.


A countryman, aged 32, apparently in good health, but rather given to drinking, and (as we learned after his death) subject to fits, was admitted 6th November, 1852, under my care, with a compound comminuted fracture of the right leg. He was riding, about half drunk, on a heavily laden waggon : in trying to get down he fell, and the wheel went over him. The skin, marked by the wheel, was injured all round the limb, but (as I hoped) not deprived of vitality. Both tibial arteries could be felt at the ankle ; and the limb was put up

in splints, without difficulty, in a good position. In three or four days it became clear that the whole of the contused skin had perished. Erysipelas afterwards appeared on the face and on the leg, spreading up to the hip. On the 18th the erysipelas was subsiding favorably: free suppuration was established from the wound, and from several deep incisions which had been made to let out the confined matter. That night he was very restless; and toward morning he kicked the bed-clothes off: the Nurse thought he had a "fit." About 3 or 4 ounces of blood were on the bed. He was in too low a state now for amputation to be justifiable, and he died quietly about noon. Several ounces of coagulated blood were found entangled with the broken pieces of bone, and injected among the muscles. The posterior tibial artery had given way by ulceration in two places, and the anterior in one place. The lower fragment of the tibia was driven firmly into the medullary cavity of the upper part.

A farm-laborer, aged 32, was admitted 21st January, 1854, under the care of Mr. Hester, with compound dislocation of the right ankle, and fracture of the outer malleolus, caused by a thrashing machine. I saw the man with Mr. Hester immediately on admission, and fully agreed with him that an attempt should be made to save the limb. I forgot to ask again after the man, and only heard by chance that he was, at his own request, removed from the Infirmary in a dying state on the 15th of March, and died two days afterwards.

The remaining cases, — 14 in number, — are added at the end of this paper: and my own opinion agrees with that of the attending Surgeon in each case, that there was no time, in any of the cases, while the patient was under treatment, in which it would have been desirable to perform amputation.

On referring to the Table of cases of Primary Amputation, it will be seen that 4 cases immediately following each other in 1853 were fatal; 2 in January, 1 in April, and 1 in May.



The symptoms observed during life were much alike in all the 4 cases ; and, in the 3 bodies which were examined after death, many similar morbid changes were noted. In the first 3 cases there was a large loss of blood *before* the operation ; yet the patients did not seem to sink from that cause. In the last case there were several attacks of hemorrhage *after* the operation ; and the man seemed to sink from exhaustion, with much the same symptoms during life as were noted in two men in 1852, who died after amputation of the thigh for disease of the knee, in both of whom there was profuse hemorrhage at the operation, or immediately after it. (183, 194.)

The circumstance that in so many fatal cases, happening about the same time, there had been a large loss of blood made us more careful about securing all the bleeding points at the time of the operation, as a means of checking what seemed one certain evil. After the chief arteries have been tied, and the tourniquet removed, every bleeding vessel has been tied with a firm thread. Since the fatal cases, — in none of which the veins were tied, — I have noted 26 cases in which the chief veins of the limb were tied ; — 13 in the thigh, in which the femoral vein, and in one the saphena also was tied, 10 in the leg, in which the popliteal, anterior and posterior tibial were tied, and 3 in the upper arm, in which the venæ comites were tied. No ill effect of any kind has been observed. In some cases, but not in all, I have observed that the ligatures on the large veins have been longer in separating than those on the large arteries.

There is not any reason to think that the result in any case was affected by the mode in which the knife was used, and the incisions made. The operation has been generally done by double circular incision, reflecting the integument before division of the muscular substance. Very few operations have been done by flaps, in cases where the soft parts were sound and admitted of circular incision.

Since the introduction of ether and chloroform, inhalation has been used at the operation.

It has been the custom to complete the dressing of the stump immediately after the operation, before the patient was taken back to bed. At one time two of the Surgeons (VII, IX) adopted the practise of leaving the wound open for several hours. In all these cases several vessels wanted tying when the wound was finally dressed ; and in many of them there was secondary hemorrhage.

The time a patient stays in the House depends on so many and such various causes, — some of them not at all connected with the operation, — and these, though often arising, are so seldom noted in the early part of the Register, that a fair estimate can not be made of the average time occupied in the healing of the wound, and the recovery of the patient.

Among the causes of delay may be mentioned the formation of abscesses after amputation of the thigh (1, 208), sinuses continuing to discharge, especially where the operation was done very near a diseased joint (224), secondary hemorrhage (193, 209, 231, 256), accidental illness, erysipelas (172, 206), smallpox (227), miscarriage (236). In 6 cases in the leg, 3 for disease (180, 236, 238), and 3 for injury (138, 170, 217), in which the knee was saved, the delay arose from the difficulty of keeping a sound covering for the stump.

Some causes of delay have been peculiar to primary amputation for injury ; such as wounds of the head and trunk, in addition to the destruction of a limb (126, 137) ; the wish to save the knee or the shoulder where laceration extended very high (137, 196) ; sloughing of skin, — in one case (in the forearm), where a flap was made of bruised integument (171½), but more commonly in the leg, after circular incision through soft parts perfectly sound, and at a

distance from the seat of injury (170, 213, 216). In one case it was followed by a tedious attack of eczema (216). Sloughing of integument has not been noted in any case after amputation for disease of a joint ; and only once in any other disease. (232.)

In one case, under my care, delay arose from an unusual circumstance. The right leg of a young man was amputated for old ulceration of eight years' standing, the result of a severe scald.¹ His left thigh had been amputated six months after the accident. After this loss of both limbs, in whatever position the poor fellow was placed in bed, he could not be kept there: he was continually shifting and rolling about, much interfering with the healing of the wound. (247).

Generally speaking, the Forearm healed rather sooner than the Upper arm, and the Upper arm rather sooner than the Leg. The Thigh is much the latest in healing. After amputation for diseased joints, the stumps healed sooner than in other diseases. The greatest delay is after primary amputation for accidents.

In reporting these cases, it has been the wish to present them to the Society as a collection of facts bearing on the question of the danger of amputation as an operation performed for the relief of suffering of various kinds. The important question of the propriety of the operation — of the fitness of the remedy for the lesion, — is not brought under notice; for that must rest in every case with the Surgeon who performs the operation.

Whether the purpose of the operation is to relieve the continued suffering caused by disease, or to save life in circumstances of urgent danger, as in accidents, the recovery of the patient — though a proof of the success of the operation in a given case, — is not necessarily the measure of success in the treatment of such cases, if the estimate is formed from those only which are submitted to operation.

¹ Museum, Ch. Ch.

The plan proposed by Mr. James, of Exeter, for arranging cases in classes of different disease, has been followed, as far as the limited number of cases would allow; and his observations on the different proportion of mortality, are to a great extent confirmed by the result of these cases.¹

Though the number of cases, compared with other published lists, is small, and though, perhaps, many circumstances which it would be desirable to know, have been omitted, — imperfect as this Analysis is, — the Compiler hopes it will not be without interest as a contribution to the Statistics of Surgery.

Postscript. — October. — As the sheets are passing through the press, the opportunity has been taken to add the notes of some cases which have come under treatment since the paper was read before the Society; and, at the same time, to include the particulars of some information lately obtained.

Cases of Injuries of the Limbs, fatal without amputation.

A feeble man, 75 years of age, was admitted 5th June, 1850, under my care, with a cut through the patella, extending into the knee-joint, inflicted by a bill-hook. No blood was lost. He gradually sank, and died within 48 hours.

A laboring man, 81 years of age, was admitted 11th June, 1850, under my care, with comminuted fracture of the lower part of the right thigh, involving the condyles, and extending into the knee-joint, and fracture of the left thigh in two places, and laceration of the skin of the leg. He was trying to get on to the shaft of a cart as it was going slowly down hill; in so doing he fell, and the wheel went over him. He gradually sank, and died on the 15th.

A laboring man, 60 years of age, was admitted 12th

¹ 'Transactions of the Provincial Med and Surg. Assoc.,' vol. xvii.

August, 1850, under the care of Mr. Hitchings, with a deep penetrating wound in the calf of the left leg, inflicted by a reaping hook. Profuse hemorrhage followed immediately, and continued till the wound was laid open, and the posterior tibial artery, and several smaller vessels, were tied; which I did in the absence of Mr. Hitchings. The man gradually sank, apparently from loss of blood, and died in 48 hours.¹

A baker, 60 years of age, was admitted 13th August, 1850, under the care of Mr. Hitchings, with his left hand and arm severely burnt, by falling into the fire during a fit. He died suddenly, in an apoplectic fit, on the 19th. Rapid decomposition followed; and the body could not be examined.

A lad, 19 years of age, was admitted in the middle of the night, 17th of January, 1851, under my care, with a pulse scarcely perceptible, all but exhausted by hemorrhage from a gunshot wound in the right popliteal space. The injury was received early in the day, 16 miles from the Infirmary, from a fowling piece, loaded with small shot, discharged accidentally within two or three feet of him. Much time was lost without any other assistance than that of a "Water-doctor," much consulted by the ignorant people of the neighbourhood. This man would not undertake the case; but late in the evening, in his capacity of Overseer (which he happened to be), he put the lad into a carriage, and brought him to the Infirmary, 14 hours after the accident. He died in 4 hours. A portion of skin, about the size of the palm of the hand, was gone, and the muscles were much lacerated. A large opening was seen in the popliteal vein; the popliteal artery was bruised, but not opened.

A half-witted man, 52 years of age, was admitted 16th of March, 1851, under my care, with a gunshot wound in the palm of the left hand, received from the accidental discharge of a fowling piece, as his hand rested on the muzzle before the shot was put in. The forefinger, middle and ring

¹ The case is fully reported in the 'Medical Gazette,' January, 1851.

fingers, became gangrenous, and were removed on the 17th of April. On the 26th, he was attacked with erysipelas of the head and face, which subsided in about a week. In the middle of May he was attacked with diarrhœa, by which he was greatly reduced. The wounds in the hand healed favorably. In the beginning of June, he began to show symptoms of increasing weakness; sloughs formed over the sacrum, and he died on the 17th. Both shoulder-joints were full of stinking pus, and the articular cartilages were ulcerated; in the left, this communicated with a large collection extending under the pectoralis major muscle. In the left wrist the cartilages were ulcerated.

A farm laborer, aged 34, was admitted 25th of November, 1851, under the care of Mr. Cleoburey, with a small unhealed wound in the palm of the left hand, between the little and ring fingers, received from an injury in a thrashing machine 4 days before admission. Some difficulty in opening the jaw was observed two or three days after admission; tetanus was fully developed on the 2d of December, and he died on the 7th.

A laborer on a railway, aged 37, was admitted late in the evening, 23d of March, 1852, under the care of Mr. Hester, with the right leg crushed by a train going over it. He was cold and almost pulseless, but sensible enough to understand the severe nature of the injury, and he gave his consent to amputation, if thought necessary. But he did not rally, and the operation was not performed. He died about 12 hours after admission.

A mason, aged 24, was admitted 19th of September, 1853, under the care of Mr. Hansard, with compound comminuted fracture of the right humerus, caused by a fall from a scaffold. Symptoms of tetanus were first observed on the 26th, and he died within 18 hours.

A laborer on a railway, aged 40, was admitted 4th of March, 1854, under the care of Mr. Symonds, with the left thigh and forearm crushed on the railway. He was sinking fast, and died in an hour.

A married woman, aged 38, was admitted 30th of

September, 1854, under the care of Mr. Hester, with destruction of the right foot and lower part of the leg, caused by a thrashing machine. She was sinking ; and she died in 6 hours.

A boy, aged 11, was admitted 19th of February, 1855, under the care of Mr. Hester, with compound comminuted fracture of the left thigh, caused by the bough of a tree striking him as it fell. He was sinking fast ; and he died in 2 hours. The femoral artery was not injured, and he had not lost much blood.

A laborer on a railway, aged 40, was admitted 20th of August, 1856, under the care of Mr. Symonds, with the right foot torn off, and the leg crushed close to the knee, on the railway. Very great hemorrhage followed. He died in 2 hours.

A married woman, about 35, was admitted 3d of September, 1856, under my care, with the right leg and lower part of the thigh torn off in a thrashing machine. She died in an hour and a half.

AMPUTATION FOR DISEASE.

Diseases of Joints.

No. in Reg.	Day of Operation.	Patient's Age.	Limb Removed.	Joint Diseased.	Day of Discharge.
1	1838	Married woman, 21.	Right thigh.	Knee, some years (V).	April 25th. Cured.
2	February 4th.	Man, 33.	Left forearm.	Wrist, some months (VI).	April 18th. Cured.
4	March 23d.	Mason, 28.	Leg.	Ankle, 6 months (VI).	May 16th. (The stump never healed: he died in about 4 years.)
7	July 27th.	Man, 53.	Thigh.	Knee, 8 months (VII).	October 9th. Died; "long-continued diarrhoea."
8	August 23d.	Lad, 17.	Leg.	Ankle, 2 years (VII).	October 24th. Cured. (Died with phthisis about 8 years afterwards.)
9	September 24th.	Girl, 17.	Right thigh.	Knee, 12 months (V).	October 24th. Cured. (Died with phthisis, 26th May 1839.)
13	December 31st. 1839	Lad, 19.	Right thigh.	Knee, 9 months (V).	February 18th. Cured.
15	January 7th.	Girl, 13.	Left upper arm.	Elbow, 2 years (VI).	February 13th. Cured. (Disease reappeared in the end of the stump, and she died in about 6 months.)
16	February 8th.	Plasterer, 28.	Right thigh.	Knee, nearly 7 years (VI).	April 3d. Cured.

18	June 14th.	Man, about 20.	Left leg.	Ankle, some months (VII).	August 21st. Cured. (Died with phthisis, in January, 1840.)
21	August 22d.	Lad, 18.	Right thigh.	Knee, many months (V).	September 18th. (The stump never healed: abscesses formed in various parts, and he died with phthisis, February 21st, 1841.)
22	June 4th.	Man, 40.	Left forearm.	Wrist, many months (VII).	June 19th. Cured. (Died with dropsy and lumbar abscess, October 2d, 1845.)
26	November 15th.	Man, 25.	Left thigh.	Knee, 10 months (VII).	February 19th. Cured.
27	November 26th.	Girl, 15.	Right thigh.	Knee, several years (IX).	April 1st. Cured. (Died with phthisis, in November, 1849.)
30	1840 April 13th.	Man, 26.	Right thigh.	Knee, several years (V).	June 3d. Made out-patient; (died with phthisis August 29th, 1840.)
36	July 13th.	Woman, 20.	Right thigh.	Knee, 2 years (VII).	October 14th. Cured.
37	July 13th. 1841	Man, 45.	Left leg.	Ankle, 5 months (VII).	September 9th. Cured.
43	January 4th.	Man, 24.	Left thigh.	Knee, many months (VII).	March 24th. Cured.
44	January 11th.	Baker, 19.	Left leg.	Ankle, 12 months (VI).	March 3d. Cured.
48	March 29th.	Woman, 21.	Left thigh.	Knee, many months (V).	May 5th. Cured.
49	April 30th.	Man, 49.	Right thigh.	Knee, many months (IX).	September 8th. Cured. (Died with phthisis, in 1843.)
51	July 12th.	Man, 29.	Left leg.	Ankle, many months (VI).	September 15th. Cured. (Died with phthisis, in 1848.)
52	August 2d.	Lad, 16.	Left leg.	Ankle, years (VII).	September 8th. Cured. (Died within a year, with scrofula. His mother died with cancer, in 1854-55.)
54	November 8th.	Lad, 18.	Thigh.	Knee, more than a year (VI).	January 12th. Cured. (Died with phthisis, 4th December, 1854.)
57	1842 January 10th.	Girl, 12.	Right thigh.	Knee, a year (VII).	April 20th. Cured. (Continued well till killed by a fall down a staircase, October, 1847.)
58	January 3d.	Girl, 11.	Thigh.	Knee, many months (VII).	February 24th. "The stump healed, but having a cough of consumptive tendency."

AMPUTATION FOR DISEASE.

Diseases of Joints.

No. in Reg.	Day of Operation.	Patient's Age.	Limb Removed.	Joint Diseased.	Day of Discharge.
1	1838 February 4th.	Married woman, 21.	Right thigh.	Knee, some years (V).	April 25th. Cured.
2	March 9th.	Man, 33.	Left forearm.	Wrist, some months (VI).	April 18th. Cured.
4	March 23d.	Mason, 28.	Leg.	Ankle, 6 months (VI).	May 16th. (The stump never healed: he died in about 4 years.)
7	July 27th.	Man, 53.	Thigh.	Knee, 8 months (VII).	October 9th. Died; "long-continued diarrhoea."
8	August 23d.	Lad, 17.	Leg.	Ankle, 2 years (VII).	October 24th. Cured. (Died with phthisis about 8 years afterwards.)
9	September 24th.	Girl, 17.	Right thigh.	Knee, 12 months (V).	October 24th. Cured. (Died with phthisis, 26th May 1839.)
13	December 31st. 1839	Lad, 19.	Right thigh.	Knee, 9 months (V).	February 13th. Cured.
15	January 7th.	Girl, 13.	Left upper arm.	Elbow, 2 years (VI).	February 13th. Cured. (Disease reappeared in the end of the stump, and she died in about 6 months.)
16	February 8th.	Plasterer, 28.	Right thigh.	Knee, nearly 7 years (VI).	April 3d. Cured.

18	June 14th.	Man, about 20.	Left leg.	Ankle, some months (VII).	August 21st. Cured. (Died with phthisis, in January, 1840.)
21	August 22d.	Lad, 18.	Right thigh.	Knee, many months (V).	September 18th. (The stump never healed; abscesses formed in various parts, and he died with phthisis, February 21st, 1841.)
22	June 4th.	Man, 40.	Left forearm.	Wrist, many months (VII).	June 19th. Cured. (Died with dropsy and lumbar abscess, October 2d, 1845.)
26	November 15th.	Man, 25.	Left thigh.	Knee, 10 months (VII).	February 19th. Cured.
27	November 26th.	Girl, 15.	Right thigh.	Knee, several years (IX).	April 1st. Cured. (Died with phthisis, in November, 1849.)
30	1840 April 13th.	Man, 26.	Right thigh.	Knee, several years (V).	June 3d. Made out-patient; (died with phthisis August 29th, 1840.)
36	July 13th.	Woman, 20.	Right thigh.	Knee, 2 years (VII).	October 14th. Cured.
37	July 13th. 1841	Man, 45.	Left leg.	Ankle, 5 months (VII).	September 9th. Cured.
43	January 4th.	Man, 24.	Left thigh.	Knee, many months (VII).	March 24th. Cured.
44	January 11th.	Baker, 19.	Left leg.	Ankle, 12 months (VI).	March 3d. Cured.
48	March 29th.	Woman, 21.	Left thigh.	Knee, many months (V).	May 5th. Cured.
49	April 30th.	Man, 49.	Right thigh.	Knee, many months (IX).	September 8th. Cured. (Died with phthisis, in 1843.)
51	July 12th.	Man, 29.	Left leg.	Ankle, many months (VI).	September 15th. Cured. (Died with phthisis, in 1848.)
52	August 2d.	Lad, 16.	Left leg.	Ankle, years (VII).	September 8th. Cured. (Died within a year, with scrofula. His mother died with cancer, in 1854-55.)
54	November 8th.	Lad, 18.	Thigh.	Knee, more than a year (VI).	January 12th. Cured. (Died with phthisis, 4th December, 1854.)
57	1842 January 10th.	Girl, 12.	Right thigh.	Knee, a year (VII).	April 20th. Cured. (Continued well till killed by a fall down a staircase, October, 1847.)
58	January 3d.	Girl, 11.	Thigh.	Knee, many months (VII).	February 24th. "The stump healed, but having a cough of consumptive tendency."

No. in Reg.	Day of Operation.	Patient's Age.	Limb Removed.	Joint Diseased.	Day of Discharge.
59	February 17th.	Man, 22.	Thigh.	Knee, many months (V).	March 24th. Died with phthisis; the stump nearly healed.
62	May 16th.	Girl, 19.	Left thigh.	Knee, many months (IX).	July 6th. Cured.
63	May 16th.	Girl, 19.	Upper arm.	Elbow, more than a year (VII).	July 6th. Cured.
69	1843 January 2d.	Man, 33.	Right forearm.	Wrist, 2 or 3 years (V).	January 25th. (Other joints became affected, and he died with phthisis, July 4th, 1843.)
70	January 16th.	Boy, 15.	Leg.	Ankle, many months (V).	May 1st. Died with phthisis; the stump having healed some weeks.
72	April 20th.	Lad, 18.	Right thigh.	Knee, many months (VI).	July 12th. (Died with phthisis 23 weeks afterwards.)
74	July 20th.	Man, 51.	Left thigh.	Knee, many months (V).	September 27th. (Died with phthisis, November 5th, 1845.)
76	September 18th.	Man, 53.	Right forearm.	Wrist, many months (V).	October 18th. Cured. (Died with phthisis, December 25th, 1846.)
77	September 25th.	Boy, 10.	Left thigh.	Knee, 12 months (VII).	November 15th. Cured.
80½	1844 March 22d.	Man, 26.	Left thigh.	Knee, years (VI).	May 1st. Cured.
81	April 26th.	Girl, 15.	Right thigh.	Knee, 2 or 3 years (VII).	June 1st. Cured.
82	May 9th.	Man, 27.	Right leg.	Ankle, some years (VI).	July 10th. Cured.
83	June 27th.	Woman, 40.	Left upper arm.	Elbow, years (VII).	July 31st. Cured. (Died with phthisis 2½ years afterwards.)
84	July 28th.	Man, 40.	Left leg.	Ankle, 20 years: slight injury 6 months (V).	September 4th. (Died with phthisis, in April, 1845.)
88	August 30th.	Man, 24.	Left thigh.	Knee, 3 years (V).	October 2d. Cured. (Died with phthisis in the spring of 1854.)
89	November 21st.	Boy, 11.	Leg.	Ankle, a year (VI).	December 18th. Cured. (Died with phthisis in about 9 months.)

92	1845 February 14th.	Man, 50.	Right leg.	Ankle, 2 years (VII).	May 7th. Cured. (Died about 6 months afterwards.) December 24th. Cured.
99	October 27th. 1846	Boy, 12.	Left thigh.	Knee, several years (IX).	February 25th. Cured. April 22d. Cured. May 27th. Cured. (Died with granular kidneys, in the Infirmary, 25th June, 1852.)
101	January 4th.	Carpenter, 48.	Left forearm.	Wrist, many months (VI).	June 24th. Cured. (After suffering from scrofulous abscesses, she was attacked with small-pox in a severe form, and died 11th December, 1854.)
103	February 9th.	Shepherd, 48.	Right thigh.	Knee, 4 years (IX).	June 10th. Cured.
104	March 9th.	Postillion, 26.	Left thigh.	Knee, many months (VII).	May 27th. Cured.
105	March 12th.	Woman, 37.	Right thigh.	Knee, about 18 weeks (VII).	June 3d. Cured.
106	April 6th.	Coachman, 24.	Right thigh.	Knee, some months (VII).	June 20th. Made out-patient with phthisis, and died in 5 weeks.
107	April 6th.	Baker, 19.	Right thigh.	Knee, about 16 months (VII).	July 22d. Made out-patient, the stump not quite healed, and died with phthisis in about 4 months.
108	April 30th.	Man, 46.	Right forearm.	Wrist, about 22 weeks (IX).	July 29th. Cured.
109	May 25th.	Boy, 14.	Right upper arm.	Elbow, 9 months (V).	August 12th. Cured.
110	June 22d.	Widow, 33.	Right thigh.	Knee, 10 months (V).	September 9th. Cured.
111	June 29th.	Woman, 19.	Right leg.	Ankle, 9 months (V).	October 21st. Cured.
112	July 6th.	Carpenter, 25.	Right leg.	Ankle, 3 years (VI).	
113	August 10th.	Married woman, 30.	Left thigh.	Knee, 8 years (VI).	
114	August 13th.	Man, 43.	Left thigh.	Knee, 21 months (VI).	
118	1847 March 29th.	Boy, 6.	Right leg.	Ankle, 8 months (IX).	June 9th. Cured.
121	June 28th.	Girl, 17.	Right leg.	Joints of tarsus 6 months (V).	August 4th. Cured.
125	November 6th.	Butcher, 50.	Right forearm.	Wrist, 9 months (X).	December 1st. Made out-patient. (Abscesses formed in his other arm, and he died with phthisis, 26th September, 1848.)

No. in Reg.	Day of Operation.	Patient's Age.	Limb Removed.	Joint Diseased.	Day of Discharge.
129	1848 January 3d.	Man, 29.	Right thigh.	Knee, 12 months (VI).	March 1st. Cured.
134	April 24th.	Carpenter, 35.	Left thigh.	Knee, 8 months (IX).	October 4th. Cured.
136	May 12th.	Man, 31.	Left forearm.	Wrist, 11 years (VI).	August 2d. Made out-patient. (The stump never healed, and he died with phthisis, in September, 1849.)
144	July 24th. 1849	Married woman, 40.	Right forearm.	Wrist, 2 years (VI).	September 1st. Cured.
149	April 2d.	Married woman, 21.	Left thigh.	Knee, 12 months (V).	May 9th. Cured. (Died with phthisis, 12th December, 1852.)
150	April 6th.	Married woman, 43.	Right thigh.	Knee, 2 years (V).	April 20th. Died, exhausted.
151	April 23d.	Girl, 15.	Left thigh.	Knee, 9 years (VI).	May 10th. Died, exhausted.
157	September 21st.	Married woman, 29.	Left upper arm.	Elbow, 9 months (XI).	October 24th. Cured. (Died with phthisis in about 2 years.)
158	November 1st.	Woman, 22.	Left thigh.	Knee, 16 months (VI).	December 26th. Cured.
159	November 5th.	Girl, 19.	Right thigh.	Knee, 10 months (XI).	November 19th. Died, exhausted : (secondary hemorrhage, November 11th).
160	November 12th. 1850	Lad, 18.	Right thigh.	Knee, 9 months (v).	May 26th. Died, diarrhoea.
167	March 21st.	Man, 35.	Right thigh.	Knee, 18 months (VI).	May 8th. Cured.
173	September. 1852	Woman, 50.	Right leg.	Ankle, 5 months (XI).	November 6th. Cured.
183	February 23d.	Man, 20.	Left thigh.	Knee, 18 months (XI).	March 17th. Died, exhausted : secondary hemorrhage.
186	March 29th.	Boy, 14.	Right thigh.	Knee, 7 years (XI).	June 9th. Cured.
194	September 13th. 1853	Sawyer, 38.	Left thigh.	Knee, 18 months (XI).	September 27th. Died. Phlebitis, pyæmia.
202	June 13th.	Girl, 18.	Left thigh.	Knee, 7 years. (Limb wasted, and hip dislocated by former disease) (XI).	July 20th. Cured. (Died with phthisis, 17th, October 1854.)

203	July 15th.	Woman, 20.	Right thigh.	Knee, between 3 and 4 years (XV).	September 14th. Cured. (Died with diabetes and phthisis, October 11th, 1855.)
208	October 7th.	Woman, 20.	Right thigh.	Knee, 6 years (XV).	January 25th. Cured.
210	December 19th. 1854	Boy, 9.	Right thigh.	Knee, 12 months (XC).	December 27th. Died, exhausted.
219	June 5th.	Man, 21.	Left thigh.	Knee, 4 years (XX).	August 9th. Cured.
221	July 14th.	Woman, 46.	Right forearm.	Wrist, 18 months (XI).	August 16th. Cured. (Died with phthisis, in August, 1855.)
223	August 7th.	Married woman, 36.	Right leg.	Ankle, 4 years (XI).	September 8th. (Died with phthisis, in the third week of October.)
224	August 21st.	Maid-servant, 21.	Right thigh.	Knee, 4 or 5 years (XV).	November 8th. Cured.
229	November 27th.	Maid-servant, 19.	Right upper arm.	Elbow, 18 months (XI).	January 17th. Cured. (The eyes and the other arm became affected, and she died with phthisis, in March, 1856.)
230	1855 March 19th.	Man, 38.	Left thigh.	Knee, 9 months (XX).	April 26th. Taken home, and died the next day with phthisis.
231	March 26th.	Butcher, 19.	Right thigh.	Knee, 9 months (XC).	May 30th. Cured.
237	June 25th.	Married woman, 31.	Left leg.	Joints of tarsus, 6 months (XC).	August 1st. Cured.
239	July 23d. 1856	Boy, 11.	Right leg.	Ankle, 3 years (XC).	August 29th. Cured.
248	March 17th.	Widow, 34.	Left thigh.	Knee, 15 years (XI).	May 21st. Cured.
249	April 7th.	Married woman, 38.	Left thigh.	Knee, 5 years (XC).	May 28th. Cured.
250	April 21st.	Glover, 25.	Left thigh.	Knee, 18 months (XV).	June 29th. Taken home, and died in two days : phthisis.
251	(Same day.)	Weaver, 68.	Left leg.	Ankle, 9 months (XV).	July 2d. Cured.
252	May 9th.	Wheelwright, 19.	Right thigh.	Knee, 21 months (XV).	July 9th. Cured.
253	May 29th.	Married woman, 32.	Left upper arm.	Elbow, 3½ years (XI).	June 25th. Cured.
254	June 16th.	Young woman, 18.	Right forearm.	Wrist, 9 months (XC).	July 26th. Made out-patient. (P.S. Stump not healed : health failing rapidly.)

AMPUTATION FOR DISEASE.

Various Diseases.

No. in Reg.	Day of Operation.	Patient's Age.	Limb Removed.	Nature of Disease.	Day of Discharge.
38	1840 July 13th.	Lad, 19.	Left thigh.	Necrosis of tibia, 4 months (VII).	September 23d. Cured.
39	August 25th.	Woman, 45.	Leg.	Caries of tibia, 2 years (V).	September 29th. Cured. (Disease appeared in the other leg and sternum, and she died in about 2 years.)
60	1842 March 18th.	Man, 47.	Left thigh.	Ulceration of cancerous appearance on the stump, about a year, after loss of the foot from gangrene many years before (V).	May 11th. Cured. (He died in about a year; "the complaint killed him.")
64	May 16th.	Man, 52.	Left leg.	Disease of tibia, following injury, many months (VII).	August 17th. Cured. (Died with some inter-nal disease, September, 1855.)

65	May 19th.	Man, 38.	Thigh.	Cancer of the bones of the leg, several years (IX).	June 17th. Died; exhaustion.
71	1843 March 23d.	Man, 23.	Left leg.	Fungoid disease of the bones of the foot, many months (VI).	June 14th. Cured.
79	November 20th.	Married woman, about 25.	Right thigh.	Caries of tibia, a year (V).	January 10th Cured. (Continued well for two years, when she died with smallpox after 3 days' illness.)
94	1845 March 31st.	Woman, 40.	Right thigh.	Caries of tibia, 17 years (V).	July 15th. Cured. (Continued well for about 7 years, when she was attacked with uterine disease and "flooding," and died exhausted, 6th February, 1853.)
117	1847 March 4th.	Girl, 16.	Right thigh.	Necrosis of tibia, without reproduction of bone, 3 years' (IX).	June 16th. Cured.
146	1848 September 19th.	Boy, 12.	Right thigh.	Necrosis of tibia, 3 months (IX).	January 17th. Cured.
165	1850 February 4th.	Porter, 35.	Left thigh.	Gangrene after ligature of femoral artery for aneurism (Nov. 26), (XI).	March 13th. Cured.
180	1851 December 15th.	Man, 48.	Left leg.	Elephantiasis, ² 14 years (VI).	February 18th. Cured. (Died with typhus fever, 9th March, 1855.)

¹ Museum St. Barth. Hosp.

² Museum, Ch. Ch.

No. in Reg.	Day of Operation.	Patient's Age.	Limb Removed.	Nature of Disease.	Day of Discharge.
193	1852 September 3d.	Butcher, 51.	Right leg.	Sinuses about ankle; with ankylosis following former disease (XI).	November 3d. Cured.
207	1853 September 16th.	Woman, 37.	Right thigh.	Useless limb, distortion of knee with ankylosis,* years (XV).	November 16th. Cured.
209	December 12th.	Man, 21.	Right thigh.	Useless limb, from paralysis, 19 years (XX).	March 1st. Cured.
225	1854 September 18th.	Boy, 14.	Left thigh.	Necrosis of tibia; — hemorrhage from perforation of posterior tibial artery (XI).	November 1st. Cured.
226 227	October 5th. October 5th.	Woman, 25. Woman, 29.	Right leg. Left leg.	Ulceration, 4 years (XI). Inconvenient stump, after amputation at the ankle-joint for injury, 13 years (XX).	January 3d. Cured. February 28th. Cured.
232	1855 March 27th.	Widow, 63.	Left leg.	Cancer of tibia, 8 months* (XV).	April 8th. Died, exhausted.
233	April 26th.	Blacksmith, 45.	Right thigh.	Ulceration of cancerous appearance, many years; with caries of the tibia (XI).	June 13th. Cured.

* Museum, Ch. Ch.

235	May 7th.	Girl, 19.	Left leg.	Caries of tibia, 5 years (XI).	July 4th. Cured.
236	May 7th.	Married woman, 29.	Right leg.	Spreading ulcers, 8 years (XG).	July 18th. Cured. (P.S. Ulceration is spreading on the stump.)
238	July 16th.	Shoemaker, 30.	Left leg.	Enlargement and suppuration of tibia, 4 years* (XV).	September 8th. Cured.
247	February 1st.	Silkweaver, 22.	Right leg.	Ulceration, after a scald, 8 years* (XV).	May 14th. Cured.
256	June 30th.	Gardener, 65.	Left leg.	Gangrene of foot, 36 days; tibial arteries impervious (XI).	October 1st. Cured.
258	July 14th.	Boy, 12.	Left thigh.	Necrosis of tibia, 3 months* (XV).	September 24th. Cured.
259	July 25th.	Carpenter, 48.	Left hand, at wrist-joint.	Diffused abscesses, sloughing of tendons, and necrosis of bones, after a punctured wound, 3 months (XV).	September 3d. Cured.
260	August 11th.	Saddler, 48.	Right leg.	Inconvenient stump, after former amputation, 12 years (XI).	(P.S. Convalescent.)

* Museum, Ch. Ch.

AMPUTATION FOR INJURIES.

Primary Operations.

(FROM THE ADMISSION BOOKS.)

Day of Operation.	Patient's Age.	Limb Removed.	Injury.	Day of Discharge.
1810 March 19th. September 2d. 1813	Man, 61. Man, 50.	Forearm. Forearm.	Gunshot wound (III). Accident (III).	May 3d. Cured. November 8th. Made out-patient.
June 11th. 1814	Young woman, 18.	Upper arm.	Torn by machinery in a mill (IV).	August 12th. Cured.
September 24th. 1820	Man, 35.	Leg.	Injury with a scythe (IV).	March 19th. Cured.
March 26th. July 29th. 1822	Lad, 16. Girl, 15.	Upper arm. Right upper arm.	Gunshot wound (IV). Torn by machinery in a mill (VI).	May 25th. Cured. October 19th. Cured.
June 13th. 1827	Man, 36.	Forearm.	Bursting of a gun (IV.)	August 1st. Cured.
January 26th.	Man, 30.	Upper arm.	Explosion by gunpowder in a quarry (V).	February 15th. Died; "inflammation of the chest."

1830 December 16th.	Boy, 10.	Forearm.	Torn by machinery in a mill (IV).	February, 10th. Cured.
1831 October 20th.	Boy, 10.	Forearm.	Torn by machinery (IV).	December 15th. Cured.
1832 May 17th.	Man, 30.	Leg.	Accident (VI).	August 2d. Cured.
1833 February 7th.	Woman, 18.	Right upper arm.	Crushed in machinery (IV).	June 20th. Cured.
1834 October 24th.	Lad, 16.	Upper arm.	Accident (V).	November 28th. Cured.
1834 December 2d.	Plasterer, 20.	Forearm.	Fall from scaffolding (VI).	January 14th. Cured.
1834 December 16th.	Man, 40.	Left forearm.	Gunshot wound (VII).	January 28th. Cured.

(FROM THE REGISTER OF OPERATIONS.)

No. in Reg.	Day of Operation.	Patient's Age.	Limb Removed.	Injury.	Day of Discharge.
24	1839 October 5th.	Woman, 55.	Left upper arm.	Torn off at the elbow by machinery (IX).	January 1st. Cured.
46	1841 March 5th.	Boy, 14.	Right forearm.	Torn by machinery (V).	March 31st. Cured.
67	1842 November 14th.	Man, 24.	Right upper arm.	Torn by machinery (V).	December 14th. Cured.
87	1844 August 17th.	Man, 47.	Thigh.	Injury to knee from a scythe (V).	September 8th. Died; gradually sank.

No. in Reg.	Day of Operation.	Patient's Age.	Limb Removed.	Injury.	Day of Discharge.
95	1845 June 14th.	Woman, about 23.	Left leg.	Foot and ankle crushed by machinery (IX).	September 24th. Cured.
102	1846 February 2d.	Man, 21.	Left forearm.	Shattered by a gun bursting (V).	February 25th. Cured.
126	1847 December 7th.	Miller, 22.	Left upper arm.	Torn by machinery (V).	January 13th. Cured.
132	1848 February 17th.	Boy, 14.	Right upper arm.	Crushed on railway (X).	April 26th. Cured.
137	May 28th.	Boy, 13.	Left upper arm.	Gunshot wound (VI).	July 19th. Cured.
138	May 29th.	Man, 35.	Left leg.	Crushed on railway (VI).	December 13th. Cured.
139	June 3d.	Boy, 14.	Right upper arm.	Compound fracture, &c., by machinery (IX).	August 30th. Cured.
140	June 5th.	Lad, 19.	Right thigh.	Crushed on railway (IX).	Died in 24 hours.
154	1849 August 1st.	Constable, 34.	Left thigh.	Leg, crushed on railway (XI).	December 13th. Cured.
162	December 21st.	Boy, 16.	Right leg.	Crushed by wagon (XI).	March 13th. Cured.
164	1850 February 1st.	Paper maker, 38.	Right upper arm.	Torn by machinery in a mill (VI).	February 4th. Died, exhausted.
170	August 2d.	Married woman, 45.	Right leg.	Destroyed in a threshing machine (XV)	October 2d. Cured.
171	August 14th.	Man, 70.	Left thigh.	Limb nearly severed by a scythe (VI).	August 16th. Died, exhausted.
171½	August 23d.	Man, 57.	Right forearm.	Torn in a threshing machine (XI).	October 30th. Cured.
172	September 10th.	Man, 30.	Left forearm.	Torn in a threshing machine (VI).	October 16th. Cured.
174	1851 March 3d.	Married woman, 54.	Right leg.	Torn in a threshing machine (VI).	May 21st. Cured.
175	May 21st.	Lad, 19.	Right forearm.	Crushed on railway (VI).	July 9th. Cured.

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176	June 13th.	Man, 45.	Left forearm.	Torn by machinery (V).	August 6th. Cured.
196	1852 December 15th.	Boy, 12.	Left upper arm.	Crushed by a cart going over it (XV).	February 16th. Cured.
197	1853 January 1st.	Boy, 14.	Left thigh.	Compound fracture, &c., in a threshing machine (XV).	January 22d. Died: pyæmia.
198	January 3d.	Man, 20.	Left upper arm.	Crushed on a railway (XV).	January 24th. Died: pyæmia.
199	April 7th.	Man, 28.	Right thigh.	Crushed on a railway (XX).	April 30th. Died: pyæmia.
200	May 13th.	Lad, 19.	Right forearm.	Torn by machinery in a mill (XI).	June 12th. Died: secondary hemorrhage, pyæmia.
204	July 18th.	Mason, 60.	Right leg.	Compound fracture, &c., by a fall from a scaffolding (XV).	September 28th. Cured.
205	August 11th.	Married woman, 52.	Right upper arm.	Torn in a threshing machine (XV).	September 28th. Cured.
206	September 10th.	Breaksman, 30.	Left leg.	Foot and ankle crushed on railway (XV).	November 9th. Cured.
213	1854 February 24th.	Man, 37.	Right leg.	Crushed on railway (XV).	May 12th. Cured.
216	March 28th.	Married woman, 54.	Right leg.	Torn in a threshing machine (XV).	June 28th. Cured.
217	April 8th.	Boy, 11.	Left leg.	Laceration in a threshing machine (XC).	June 14th. Cured.
261	1856 August 24th.	Boy, 12.	Left forearm.	Gunshot wound (XC).	October 1st. Cured.
262	October 2d.	Man, 31.	Left leg.	Crushed in a threshing machine (XC).	

AMPUTATION FOR INJURIES.
Secondary Operations.

No. in Reg.	Day of Operation.	Patient's Age.	Limb Removed.	Nature of Injury.	Day of Discharge.
20	1839 July 23d.	Postillion, about 55.	Thigh.	Mortification following compound comminuted fracture of leg, July 15th (VII).	September 11th. Cured.
122	1847 June 28th.	Boy, 13.	Right forearm.	Sloughing after laceration by machinery, June 22d (V).	July 21 st . Cured.
153	1849 May 22d.	Gamekeeper, 41.	Right leg.	Gunshot wound, May 1st. Admitted May 3d (VI).	July 4th. Cured.
155	August 23d.	Man, 26.	Left thigh.	Gangrene following fracture of the thigh, July 27th, admitted 28th: rupture of popliteal artery and vein (XI).	October 24th. Cured.
169	1850 June 24th.	Lad, 19.	Right leg.	Sloughing after injury on railway, January 1st (V).	August 7th. Cured.
234	1855 April 30th.	Married woman, 45.	Right upper arm at shoulder-joint.	Scald or burn, April 21st, admitted 24th (XV).	May 1st. Died; maniacal exhaustion.

ON THE
STRUCTURE AND NATURE OF THE SO-CALLED
COLLOID CANCER.

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ALTHOUGH the subject of colloid cancer has attracted considerable attention from pathologists, many points in regard to the structure and nature of the disease remain in obscurity. The following account will be found to differ in several particulars from the descriptions usually given, which do not, I think, convey a perfectly satisfactory idea of the peculiarities of the disease. The fact of the association of a membrane in and with the stroma of colloid has been indicated by several authors; but I am not aware of any account of the disease, in which the characters and development of this membranous stroma have been fully described. The description, too, of the contents of the stroma will be found to differ somewhat from that which has hitherto been given.

The names colloid, and colloid cancer have been applied to a great variety of pathological conditions; and it is probable

that many diseases having no relation to one another have been thus classed together. Some writers, and especially those of Germany, apply the term colloid to almost any thick fluid, the name alveolar cancer being given by them to those diseases which we commonly call colloid cancer. It is well known that almost every grade of transitional formation is met with, from the colloid cancer, as seen in the stomach, to the thick adhesive serum of some cysts, and it may sometimes be difficult to state with certainty to which class a particular case belongs. In the following communication it is therefore proposed to restrict the descriptions to unequivocal examples of colloid or alveolar cancer. I have observed nine examples of this disease, and, in addition to these, specimens have been examined from other cases furnished me by friends, besides a considerable number of those colloid diseases which are generally considered not to be cancerous; such as occur in the thyroid and in other parts of the body.

Although the microscopical appearances of colloid may vary in different cases, almost as much as the naked eye characters, yet as a general type of structure is found to prevail, this type will be first described, and then the various aberrations from it.

Colloid cancer, as seen by the unassisted eye, is obviously composed of a fibrous looking stroma supporting a quantity of gum-like material (fig. 1). The proportion in which these elements are combined varies extremely; indeed, it appears that in some cases the stroma may be nearly absent, and then the disease assumes the form of an ill-made jelly; on the other hand, it may be so abundant that the mass is as firm as a cartilaginous tumour.

Colloid is essentially an infiltrating disease, i. e., its elements are produced amongst a pre-existing tissue; and so in a section of colloid, portions of the structure which had become the seat of the disease are generally detected. Good examples of this condition are frequently seen in the stomach, where muscular fibre and areolar tissue may be observed mixed up with the morbid product (fig. 8).



It is probable that all the fibrous structure, which is similar to normal areolar tissue, belongs to the organ or part infiltrated, and not to the new growth. This view is chiefly borne out by the fact that areolar tissue is completely absent in the looser and possibly purer portions of colloid, and that it is most abundant where there is to the eye the greatest amount of the original tissue remaining.

The stroma of colloid, in its most characteristic form, assumes the shape of a convoluted membrane. In many places it is entirely composed of this tissue, when it then appears like the lining membrane of a multilocular cyst, such as occur in the kidney or ovary. The membrane is simple, thin, finely granular, with occasional markings, chiefly in straight lines, upon it (figs. 2, 7). In places it is so thin and transparent that its presence may be overlooked. The markings or lines upon this membrane are wanting on the smooth portions; but are extremely abundant where it is folded or creased. In such parts the lines correspond to the folds of the membrane, and spread out in a fan-shaped form from the twisted portion. In places the stroma has a fibrous appearance, which in some instances is obviously owing to the folding and stretching of the membrane; in others this structure is probably due to the membrane having split up into fibres in a direction corresponding to the folds.

In many places, however, well-defined fibres, presenting peculiar characters, are seen; they are more or less flat or ribbon-shaped, extremely thin and transparent, generally faintly, but sometimes coarsely, granular. No nuclei can be discovered among them. Here and there they expand out and are continuous with the membranous stroma already described. In some places they are thin and filamentous; but in others they resemble the yellow fibres of areolar tissue. The above similarity in texture, and the frequent junctions of these fibres with the membrane (fig. 4), indicates that they are parts of the same tissue.

Although the stroma of colloid is generally described as being entirely fibrous, some authors have recognised the presence of a membrane associated with these fibres.

Rokitansky¹ has described a membranous form of stroma in cancer, which he says is strongly marked in the colloid variety; the arrangement he compares to a trellis-work intervening the colloid material. Mr. Paget, in his lectures, speaks of the stroma of colloid as formed by "fibres or fibred membranes," elongated nuclei being frequently seen among them.

It is thus seen that the stroma divides the colloid substance into loculi. These may be sub-divided again and again into still smaller spaces by similar but more delicate stromal tissue.

The size of the loculi thus formed is extremely variable, the smallest being little larger than a blood-corpuscle, the largest the size of a hen's egg. Between these extremes every possible variety in size may occur, and this fact constitutes one of the most remarkable features of the disease.

The spaces enclosed by this membranous stroma are filled with the essential element of the disease, that is with colloid material. This is arranged in more or less globular masses, or in a cluster of such masses (fig. 5). They may easily be separated from the stroma either by pressure or by scraping the surface of a section. The bodies which are thus separated are similar in shape and size to the loculi from which they have been withdrawn. The fact of their becoming displaced so readily appears to indicate the existence of a complete partition between many loculi where no line of separation can be detected by the eye.

These rounded masses of gelatiniform substance, which are termed colloid bodies or corpuscles, may be composed of a central body or kernel surrounded by gelatinous substance, or may be wholly made up of the latter material.

The central portion or kernel may be nearly spherical or ovoid, or it may appear as if splitting up into several parts (fig. 11). It is often composed of a large number of simple spherical cells closely packed together, each of which, in many cases, appears to contain a large spherical nucleus;

¹ Rokitansky, 'Path. Anat.,' vol. i; and his Essays on 'Cancer stromata' and 'Colloid Cancer,' quoted in the above, Syd. Soc. Edit.

in some instances indications of a cell-wall surrounding these may be seen. In other examples the cells composing the kernel are less definite in form, and may be represented only by an aggregation of granules, probably phosphate or carbonate of lime.

The jelly-like substance surrounds the central kernel with a certain degree of uniformity in all directions, like the soft part around the stone of a fruit. It is elastic, and on being indented, readily regains its former shape. Occasionally, concentric lines may be detected. If, however, the concentric arrangement is not obvious in the natural condition of the structure, slight pressure, or the addition of a little iodine or acetic acid will render it apparent. Granules, and, occasionally, cells, will also be seen arranged in these concentric lines. A little examination will soon prove that the lines are not fibres, but that the appearance is due to the substance of which the "colloid body" is composed, having a concentric arrangement, similar to the coats of an onion. This may be conclusively demonstrated by placing one of the colloid bodies beneath a dissecting microscope, and turning it over with a needle, whilst still in view.

In many specimens, it appears as if some of the granules lying between these concentric laminæ, had become the nucleus or kernel of a new colloid body (fig. 6), thus forming a compound colloid corpuscle (figs. 9, 12).

Moreover, fresh corpuscles may arise in the interior of younger ones, and in this manner, endogenous growth may take place to almost any extent (fig. 6).

Frequently, however, there is but little evidence of a central body or kernel to the colloid corpuscle. It then appears as a more or less spherical mass of gelatinous substance, having generally a laminated arrangement, granules and cells being scattered throughout its interior. The cells so placed are subject to irregularities both in form and size. Some are as small as blood-corpuscles, whilst others approach the magnitude of ordinary colloid bodies; suggesting to the observer that the smaller, supposing development to continue, would gradually attain the

dimensions of the larger bodies ; in other words, that they would become ordinary colloid corpuscles.

Occasionally, the smaller cells of colloid are very numerous, and sometimes occur in great numbers near the surface of the colloid body, and thus appear to form an epithelial lining to the membranous stroma.

It is commonly stated that when acetic acid is added to a section, nuclei are seen in the stroma. On careful examination I have not been able to detect either cell or nuclei among the stromal tissue. I am inclined to believe that the structures which are rendered apparent in the gelatinous substance by the addition of acetic acid, have been mistaken for, and described as stroma containing nuclei. The effect, however, of the reagent is to obscure the stroma, and this result is produced not so much by any action on that tissue itself, as by the acid rendering strongly conspicuous the laminated arrangement of the transparent portion of the mass, which thus marks the intervening stroma. It is possible that many of those bodies resembling oat-shaped nuclei, which are seen in the gelatinous portion of the corpuscles, are only separations or spaces between the laminae.

In a case of gelatiniform cancer of bone, Wedl¹ has observed the laminated arrangement of the colloid substance around the central mass. The same author noticed that, on the addition of acetic acid, both in this case and in one of colloid of the breast, bodies like elongated nuclei were seen arranged in concentric lines. He considers that this appearance was caused by the action of the acetic acid, and that the elongated bodies were 'coagulating mucin.' The stroma in these cases is described as fibrous.

The description that has been given applies to all the more dense forms of colloid cancer, but where the disease assumes the appearance of a simple jelly, many of these characters are lost. As Dr. Jenner has very accurately described the structure of this substance in a case he brought

¹ 'Path. Histology.'



before the Pathological Society¹ I need only very shortly recite its characters.

The transparent jelly-like substance from Case 5, when examined under the microscope, is composed of an homogeneous material, in which some cells and a few granules are embedded. Of these cells, a few are transparent, spherical, and about the size of pus-globules, whilst others are larger, and filled with granules like compound granular corpuscles.

By the addition of acetic acid, it becomes opaque, and extremely tough. If now placed under the microscope, it exhibits a somewhat indistinct appearance of parallel striæ, the granules and cells before mentioned being arranged with their long diameters in the direction of these streaks. It is evident that this structure does not indicate the existence of fibres, but resembles the streaking which may be seen in mucus after the addition of acetic acid.

The opaque blanc-mange-like substance, from the same case, appears to be almost entirely formed of dark, spherical granules, and some larger angular bodies, embedded in a transparent matrix. As was originally described by Dr. Jenner, these granules very closely resemble those of fat; both these and the larger bodies are dissolved by hydrochloric acid, the latter leaving an animal basis without change of form. Some are dissolved without obvious action, but in the majority, a brisk effervescence may be noticed. The acid which was added is now found to have become impregnated with phosphoric acid.

An unusual and very remarkable structure was observed in one of the cysts from the same case (No. 5). It was about the size of a hazel nut, and did not present any external characters by which it could be distinguished from its neighbours. When cut into, a transparent jelly-like substance protruded, in which an immense number of little sacs were embedded. The small sacs or cysts were mostly spherical, and rather more opaque than the surrounding material; in size they varied from that of a blood-corpuscle

¹ 'Trans. of the Path. Soc.,' vol. v.

up to that of a hempseed. They were composed of a membrane enclosing a globular mass of jelly-like substance (fig. 9). The membrane was coarsely granular, easily torn, and of some thickness. Smaller cysts were often enclosed in the large ones. Large numbers of granules and cells (fig. 11), such as are usually met with in colloid were seen in all parts of the jelly-like substance. Cholesterine, which is seldom absent in colloid, was very abundant in this example.

Although at first sight the structure of this particular cyst appears distinct from that of the firmer forms of colloid, the difference is only in the degree of consistence of the various portions of the disease. As a general rule in firm colloid, the density of the stroma is not greatly different from that of the contained substance. If, however, we imagine a compound colloid body, in which the membrane both of the parent and of the enclosed corpuscles were thick and tough, whilst the contents were somewhat looser than usual, the structure would be precisely that of the cyst described. This cyst may, therefore, be regarded as a compound colloid body.

The free jelly-like substance may be explained as the result of an extreme development of colloid substance, with scarcely any stroma. It is not improbable that the comparative absence of stroma may be occasioned by the rupture of some of the cysts, and the continued secretion of colloid material from their walls.

Development.—The mode of origin of colloid is far less easy to trace than its manner of increase. The disease, in its most elementary form, probably exists as a simple cell. The colloid cell appears to be developed around certain granules or nuclei, which are found in an otherwise healthy tissue near the disease, and may or may not be previously aggregated into a mass.

The endogenous mode of growth of these colloid bodies is as follows; the kernel frequently exhibits a tendency to split up into a number of separate masses (fig. 10), each of



which may become the kernel of a fresh colloid body. In some instances a few nuclei or even granules become detached from the surface of the kernel (fig. 6), and form a fresh one. In this manner, a compound colloid corpuscle is produced; the process may, moreover, be repeated again and again.

The fact that the kernels of many if not of the majority of the colloid corpuscles are composed of granules, tends to disprove the hypothesis which suggests itself, viz., that these granules are simply degenerated nuclei.

A number of colloid bodies arising and increasing in the manner indicated, become blended together into a mass, and the cell-walls of the colloid bodies become partially absorbed. This process is traced with greater or less distinctness in different cases, but was extremely obvious in a case of colloid of the peritoneum (fig. 9).

The stroma of the disease is thus formed by the coalescence of the walls of the colloid bodies, and hence its close similarity to the lining membrane of a multilocular cyst. The peculiar ribbon-shaped fibres which are seen spreading out into portions of membrane, are no doubt the remaining portions of the same tissue, the rest having been absorbed (fig. 4).

The character of the fibres of the stroma, as well as the complete absence of nuclei among them, indicates a different nature and mode of growth from that of ordinary areolar tissue. Most of these fibres are produced by the splitting up of the membrane, perhaps some may be formed by the coagulation of a fibrinous blastema.

It is difficult to account for the peculiar lamination of the colloid material. The nearest analogy in structure is probably that which is described to exist in the vitreous humour. It is possible that the endosmosis by which the colloid cells become distended goes on at irregular rates, or perhaps with intermissions, so that one lamina becomes as it were consolidated before the next one is formed over it. Another hypothesis which suggests itself is, that the arrangement is produced by a process similar to that by which

fibrinous concretions are formed in the peritoneal cavity,¹ i. e., by the deposition of the material, lamina after lamina, on a central body or kernel.

Relation to cancer and other diseases.—The want of uniformity of opinion as regards the relationship of cancer and colloid, to a certain extent depends on the variety of diseases that have been included under the name colloid. Formerly colloid, in all its forms, was classified with malignant diseases, but it is now more generally taught that it may assume a non-malignant or a malignant character; and it is the latter class only which is under consideration.

In order to determine the claim of the so-called colloid or alveolar cancer to be placed among the cancerous diseases, it will be well to bear in mind what are usually admitted to be the essential characters of the latter disease, viz. :

1. Its structure and chemical nature.
2. Its infiltration among the natural tissues of the body.
3. Its invasion of the lymphatic vessels and glands.
4. Its tendency to destruction by ulceration and sloughing.
5. The occurrence of secondary growths, particularly in the lungs and liver.
6. Its tendency to recurrence after removal by operation.
7. The presence of a peculiar cachectic condition of the system.

Although one or two of these conditions may be absent in individual examples of the disease, still a group of cases in which the same character or characters are uniformly absent cannot with propriety be called cancer.

As to structure, the arrangement of colloid that has been described is altogether dissimilar from the ordinary forms of

¹ 'Path. Trans.,' vol. vi.

scirrhus, medullary, or epithelial cancer. Bodies resembling cancer-cells, are, it is true, not infrequently met with in colloid, but careful examination will generally prove them to be growing colloid-cells. So also chemical analysis of colloid shows it to be a substance perfectly *sui generis*.

With regard to the affection of the lymphatic glands, the only way in which they appear to become involved is by the extension of the disease to them, and thereby enclosing them in the tumour. There does not appear to be any evidence of their proneness to become affected with the disease rather than the other tissues.

As to secondary growths, it will be seen that, although in several cases there was more than one example of colloid disease in the same body, in none were these tumours in the substance of the liver, or in that of the lungs. Moreover, I cannot refer to any completely recorded example in which growths were discovered in the substance of those organs,¹ still there are several well authenticated cases, in addition to those appended, in which more than one colloid tumour was present in the same individual.

In those cases in which several organs were involved, it is obvious that this peculiarity arose from the extension of the original disease, its almost complete continuity having been traced.

It is also seen that in all the examples of multiple colloid tumours the disease was confined to the abdominal cavity, and not only to this but to two parts of it, the peritoneum and the intestinal canal. The manner in which the intestines were affected is strikingly peculiar; colloid growth taking place at isolated portions of the muscular coat of the

¹ The condition of the lungs in the very interesting case given by Dr. Warren ('Med.-Chir. Trans.,' vol. xxvii), may appear an exception to this rule. The loss of transparency which occurred in these tumours on being kept in spirit, renders their colloid nature very doubtful; so that in the absence of microscopical description it cannot be considered as perfectly proved that these were really examples of the so-called colloid cancer.

bowel, as in Case 4.¹ Of the manner in which the disease spreads over the surface of the peritoneum, Case 5 furnishes a good example.

This strict limitation of the disease contrasts strongly with the ubiquitous character of cancer, from the invasion of which no tissue and no part of the body is free.

Without entering into the question of the value of cachexia as a distinctive character of cancer, I believe there is no proof of the existence of any special cachexia in colloid, beyond that induced by the presence of serious and fatal disease.

May colloid be associated in the same growth with cancer? The appended cases do not furnish an example of the coincidence of colloid and cancer. There does not, however, appear, *à priori*, to be any reason for believing that the two might not sometimes be associated in the same way that cancer occurs with cystic disease.

Occasionally an apparent combination of colloid and encephaloid cancer is seen in the stomach; that is to say, there is a cancerous ulcer in some part of the stomach, with thickening near the pylorus, which somewhat resembles that produced by the so-termed alveolar cancer. I have carefully examined two examples in which these appearances were noticed; but in both the thickening near the pylorus depended solely on hypertrophy of the muscular coat.

Again, certain tumours of the firmer forms of medullary cancer sometimes exhibit an apparent combination of colloid and cancer; that is, portions of these tumours are singularly clear and translucent, closely resembling in this respect colloid cancer. In all such cases, however, that I have examined, the characteristic structure of colloid is wanting.

Relations of colloid to tubercle.—Whilst the indications of the association of colloid with cancer are so slight, the large proportion of cases in which it was associated with tubercle is striking. In some examples the tubercle appears not only of recent formation, but to have been in a state of

¹ See also 'Guy's Hospital Reports,' Series III, vol. i, where a somewhat similar case is recorded.

activity. When bringing forward an example of the concurrent development of cancer and tubercle,¹ these cases were not adduced, as they were not considered true examples of cancer.

The foregoing leads me to the conclusion that, as far as can be judged from the cases that have fallen under my observation, colloid is a disease perfectly *sui generis*, and is neither of a cancerous nature nor frequently associated with cancer. The cases, moreover, on which this conclusion is chiefly founded are examples of several of the most characteristic forms of the first-mentioned disease.

Little can be said regarding the treatment of colloid. When within reach of the knife there can scarcely be a doubt as to the propriety of removing the tumour, for although the cases of external colloid before the profession are not sufficiently numerous to decide the question, they show that the disease, if not radically cured, is at least slow in returning.

In concluding this paper, I have to thank my friend, Mr. Flower, for the accompanying drawings, which very faithfully represent the microscopical appearances of the disease.

APPENDIX.

The following is an abstract of the cases mentioned in the foregoing paper. With the exception of Case 2, they were all patients of the Middlesex Hospital :

CASE 1.—A female servant, unmarried, æt. 21,² was

¹ 'Med. Times and Gazette,' 1853.

² The ages given are those of the patients at the time of attack, or when the disease was first discovered.

admitted, August, 1852. She had suffered from pain and difficulty of defecation for twelve months. A stricture of the rectum was discovered, which resisted the usual treatment. She died somewhat suddenly, from a perforating ulcer of the colon, after the disease had existed for fourteen months.


Post-mortem.—The rectum, from ten inches above the anus to the sigmoid flexure, was converted into firm "colloid cancer," which, in many places, was two inches in thickness. The other viscera were healthy. (See Museum of Middlesex Hospital).

CASE 2.—A tumour formed in the breast of a lady, æt. 45. It increased gradually and without pain, till it attained the size of a walnut, and was then (November, 1851) excised by Mr. O. Clayton. The tumour, on section, presented the ordinary appearance of firm colloid cancer. The wound healed rapidly after the operation. The patient remains perfectly well at the present time (March, 1856). The scar is healthy.

CASE 3.—A swelling made its appearance in the right parotid region of a woman, æt. 61. This slowly increased for six years, till, in February, 1853, it was excised by Mr. Shaw. The tumour was then as large as a goose's egg, and exhibited the usual appearance of colloid cancer. The wound healed, and she was perfectly well when seen last, about the end of 1855.

CASE 4.—A man, æt. 40, had suffered for six months before his death with pain after food, constipation, and vomiting. He died, May, 1854, with symptoms of obstruction of the bowels.

Post-mortem.—The pyloric end of the stomach was converted into firm "colloid cancer" half an inch in thickness. There was similar disease at the lower end of the ileum, and also in the colon. There were numerous crude tubercles in the lungs, on the pleura, and on the peritoncum.



CASE 5.—A man, æt. 43, died, in January, 1854. He had suffered from “coldness” in the stomach, vomiting, and swelling of the abdomen, for six months.

Post-mortem.—The whole of the abdominal viscera were completely bound together by colloid cysts and colloid material. There was also about a pint and a half of free colloid, resembling ill-made jelly. A portion of the disease was as firm as enchondroma. The various viscera were surrounded, but not invaded by the growth. (See Museum of Middlesex Hospital.) The lungs were infiltrated throughout with miliary tubercle, which presented the usual structure under the microscope.

CASE 6.—A man, æt. 40, was attacked with dyspepsia, particularly after taking animal food. Soon after this he suffered from pyrosis, and died, May, 1855, six months after the first symptoms of disease.

Post-mortem.—The stomach retained its natural form; but was converted into a hard unyielding sac. The walls being infiltrated with “colloid cancer” and being nearly five eighths of an inch in thickness. There were some old and recent tubercles in both lungs.

CASE 7.—A man, æt. 40, discovered a tumour, about the size of a walnut, in his right iliac region. This slowly increased till he died, December, 1854, five years afterwards. He had suffered principally from irritability of the bowels and the frequent passage of blood.

Post-mortem.—A “colloid” constriction of the ascending colon, with infiltration of the neighbouring bowel.

CASE 8.—A man, æt. 63, suffered from diarrhœa and occasionally from vomiting, for seven months, and died, May, 1855.

Post-mortem.—A tight constriction of the transverse colon was discovered, with deposit of colloid material in the walls of the bowel, and principally in the muscular coat. The colon a short distance below, was affected with similar disease.

CASE 9.—A tumour formed in the right breast of a female mulatto, æt. about 35. This slowly increased for twelve months, when the breast was excised by Mr. Moore, December, 1855. The tumour was then about the size of a hen's egg, and was composed of hard, translucent colloid. The wound healed rapidly, and she left the hospital apparently well.

DESCRIPTION OF THE PLATES.

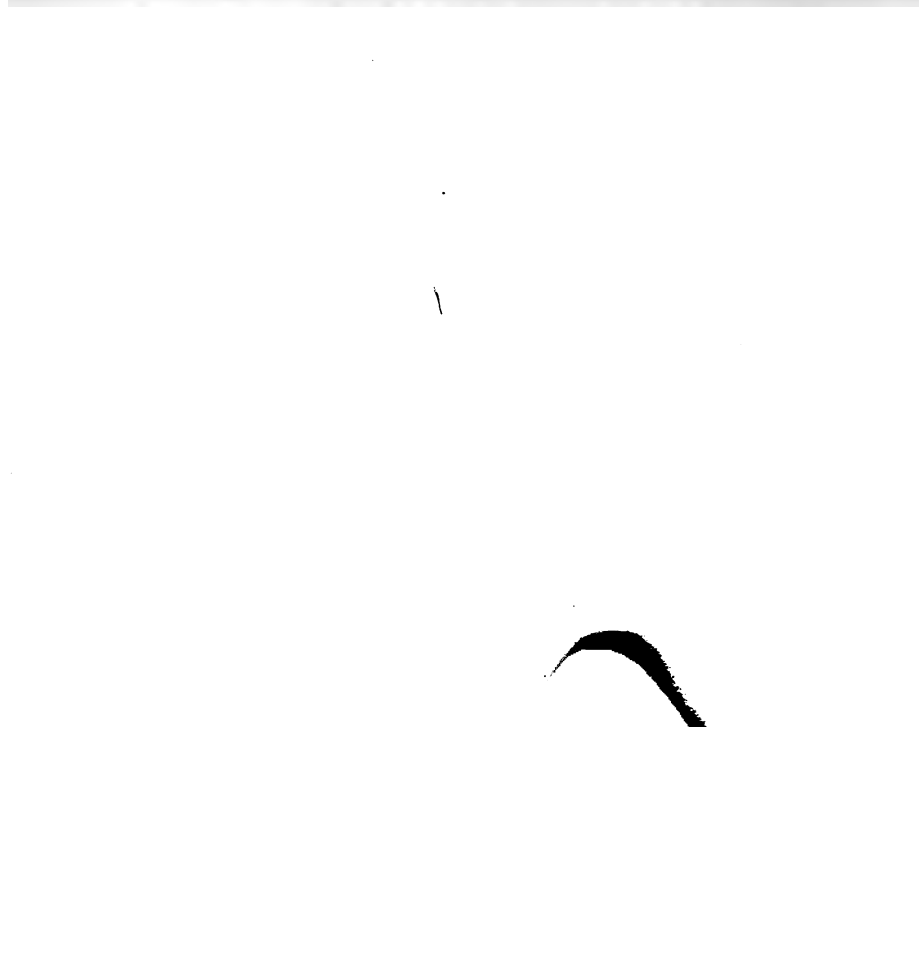
PLATE I.

Fig.

1. Section of colloid of the breast (Case 8).
2. Section of colloid of the stomach (Case 6).
3. Section of colloid of the rectum (Case 1).
4. Fibrils and stroma of colloid of the breast (Case 8).
5. Stroma of colloid of the peritoneum (Case 5).
6. Section of colloid of the breast, to which acetic acid has been added (Case 8).

PLATE II.

7. The membranous stroma from colloid of the breast.
8. The muscular coat of the stomach invaded with colloid.
9. Development and fusion of the colloid cysts from the peritoneum (Case 5).
10. Simple colloid bodies.
 - a.* In the natural condition.
 - b.* After the addition of iodine.
 - c.* After the addition of acetic acid.
11. Kernels of the colloid bodies (Case 8).
12. Compound colloid body (Case 1).



C A S E
OF
FRACTURE OF FOUR CERVICAL VERTEBRÆ
WITH DISLOCATION,
PRODUCED BY SLIGHT AND UNUSUAL CAUSE, AND RESULTING
IN IMMEDIATE DEATH;
WITH
NOTES OF A CASE OF FRACTURE OF THE OS CALCIS.

BY
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COMMUNICATED BY
SPENCER SMITH, Hon. Sec.

Received April 19th.—Read April 23d, 1856.

ON the night of Sunday, October 28th, 1855, a man was brought to the Hospital in a cab; and, on examination, was found to be dead. The men who brought him gave the following account: A number of friends were drinking together in a public-house, when the deceased, in sport, snatched a hat from the head of another man, and placed it upon his own, calling out for somebody to "bonnet" him. Five or six blows were struck with the open hand on the crown of the hat, when he rushed at one of the party and butted him in the chest. This man seized the hat by the

brim and forcibly twisted the head from side to side several times, pushing the deceased back upon a form in the sitting posture. His head then dropped forwards, the chin resting on the chest, and, muttering "Fetch a doctor," he slipped off the seat upon his knees, and would have fallen upon his face had not some of the bystanders caught him in their arms. They took him directly to a surgeon in the neighbourhood; but, beyond a groan, the injured man gave no evidence of life. He was brought to the Hospital about forty minutes after the accident; all the party were quite sober.

The body, awaiting an inquest, was not examined until the fourth day after death. The head and neck were preternaturally moveable; but, beyond a graze of the skin upon the point of the chin, there was no external mark of injury.

Between the deep-seated muscles attached to the upper cervical vertebræ a small quantity of blood was extravasated. There was fracture of the posterior arch of the atlas, also through the laminae of the second, third, and fourth cervical vertebræ directly behind the articulating processes, completely separating them from their respective bodies, in addition to which the laminae of the third and fourth on the left side were comminuted. The line of fracture passed through the bones obliquely from above downwards and forwards. The third vertebra was partially dislocated forwards with its right inferior articulating process resting in the groove for the fourth spinal nerve. The corresponding process of the left side was displaced upwards and rather forwards, both the capsular ligaments being partially torn through. The apices of these processes were broken off, and remained attached to the laminae on either side. The point of the superior articulating process of the fourth vertebra was also broken off on the right side.

The posterior atlo-axoid ligament was much bruised, in parts almost disorganized. The ligamentum subflavum between the second and third vertebræ was torn away from the lamina of the second.

The areolar tissue occupying the space between the bones



and the membranes of the cord, from the first to the fifth vertebra inclusive, was filled with coagulated blood. The theca vertebralis did not contain any blood. The spinal cord was not flattened; but opposite the third vertebra it was bruised, blood being effused into the substance of the anterior and lateral columns.

The posterior common ligament was much stretched, with a longitudinal rupture, to the left of the median line about an inch in length opposite the second and third vertebræ, through which a mass looking like coagulum protruded. This, on examination, proved to be intervertebral substance from between the third and fourth vertebræ, which was crushed and mixed with small scales of bone. A probe could be passed through this opening in the ligament downwards behind the body of the third vertebra into the space left behind it and the fourth. The odontoid process with the alar and transverse ligaments was uninjured. The anterior common ligament was entire but tensely stretched.

The brain and medulla oblongata presented no lesion.

The bones appeared to be quite healthy, and the manipulation necessary to their preservation has since shown them to be so.

On reviewing this case in all its bearings, the extreme importance of it, in a medico-legal point of view, cannot fail to strike the observer; for, had not the small amount of violence which was used been witnessed by so many persons, it would scarcely have been believed that fracture, with dislocation, could possibly be produced by such a trifling accident.

To satisfy myself that the statement which I had received was correct, I took the trouble, five or six weeks afterwards, when all motive for concealment was at an end, to call upon six of the men separately, and to inquire again into the manner of the accident; but they completely corroborated the statement before given. We must, therefore, endeavour to account for so much mischief by other causes than a mere blow with the open hand.

The position in which the spine would be bent with relation to the head, the latter being then thrust against an opposing body, together with the corresponding *contre-coup* will, I think, satisfactorily account for the injury. The spinal column being semiflexed, with the head thrown backwards upon it (as must have been the case when the man was bent in a butting attitude, endeavouring at the same time to keep his hat from falling off), the head with the two upper cervical vertebræ would have an axis, which would meet the rest of the spinal column at an acute angle, the apex of it centering in the third vertebra. At the same time the bodies of the cervical vertebræ would be separated from one another as widely as possible, and the spinous processes made to press upon each other firmly. If, while in this position, the man rushed forward and brought his head in contact with an opposing object, force would be applied by *contre-coup* in a direction opposite to that of the body, and the consequence would be the meeting in the third cervical vertebra of the two forces: that communicated through the head and two upper vertebræ, would force the third vertebra downwards and backwards, while that communicated through the rest of the spinal column would press it downwards and forwards. If these forces met at a right angle no doubt the body of the bone would be crushed; but if, as I presume in this case, they met at an acute angle, the pressure would be communicated to the articulating surfaces and spines, the articulating surfaces would give way, and the vertebra slipping forwards, the weight of the body would be suddenly thrown upon the spinous processes, and such forcible compression be produced as to cause the fracture of them. The ligamentum subflavum would at the same time be ruptured, while the laminæ of the third and fourth vertebræ on the left side would be comminuted, from being made to press against each other, in consequence of the displacement described; the third on the right side being internal to that of the fourth would escape pressure.

The space between the arches of the vertebræ and the cord, being in this region considerable, there would be



sufficient room for the medulla to pass without suffering compression from such a disarrangement of parts; but the plexuses of veins, which ramify so plentifully upon the vertebræ within the canal, would be lacerated, and death would ensue from pressure upon the spinal cord.

Such was the state of parts found, and I can only, satisfactorily to myself, explain this injury in the manner above described.¹

This opinion with respect to the mode of accident is corroborated by Malgaigne, where he states that *contre-coup*, with a forced flexion of the spinal column, is by far the most common cause of fracture.²

Dupuytren, in his list of cases, twenty-eight in number, does not record any at all analogous to this; Malgaigne mentions nothing similar as occurring in his own practice; but he refers to a case cited by Reveillon, in which a transverse fracture of the body of the fifth cervical vertebra was occasioned by a sudden and forcible extension of the neck when the head was already bent backwards.³ Ollivier has collected many cases, but none parallel to this.

There is an instance, related by M. Lasalle, in the 'Gazette Médicale,' of November 21st, 1841, where by a sudden and forcible extension and flexion of the head, death immediately resulted, the ligaments being ruptured, the intervertebral substance torn through, and the vertebræ separated, with effusion of blood into the theca vertebralis. I can find no cases recorded by English surgical writers but such as were occasioned by external violence directly applied.

It is manifest that the injury, in the case which forms the subject of this communication, occurred in no ordinary

¹ It is also possible that death might have been occasioned by compression of the cord by the fractured bones, which had afterwards changed their position in consequence of the various movements to which the body was subjected after death.

² Vide 'Traité des Fractures et des Luxations,' par J. F. Malgaigne, tome i, pp. 418-19-20.

³ This case is also mentioned by South, in his translation of Chelius' 'System of Surgery,' vol. i, p. 533.

manner, from the fact that it was the third vertebra which was displaced, and which, according to Malgaigne's experience, is (with the exception of the atlas) the least liable to dislocation. He records forty-one cases of luxation in the cervical region, and in a single instance only was the third vertebra removed from its position, and then but partially: this experience of Malgaigne is confirmed by all the recorded cases.

The rarity, then, of luxation of the third cervical vertebra being established, it may be presumed that it would require extraordinary causes to produce it, and would be attended with greater injury to other parts than any more common displacement.

NOTES

OF A

CASE OF FRACTURE OF THE OS CALCIS.

BY THE SAME AUTHOR.

Thomas Bassett, æt. 44, stableman, admitted into St. Mary's Hospital, September 19th, 1855, under the care of Mr. Ure. This afternoon, about an hour before admission, he jumped from the wheel of a carriage, alighting upon his heel; this gave way under him and he fell, but arose directly, experiencing great pain, and inability to put the sole of his foot to the ground; he walked, however, into the hospital, from the street, with assistance. There was some swelling and ecchymosis, but no distortion of the foot. On examination, there was found to be unnatural mobility of



the heel, and crepitus was obtained by moving it laterally, as well as by holding the heel and flexing the ankle; when the sides of the foot were grasped firmly, embracing the malleoli, and the patient moved his toes, crepitus was also produced. When told to walk, he could bear his weight partially upon the toes, but a loud snap was heard, and he felt a grating at the heel; on pressing the calf of the leg, or irritating the gastrocnemius, this was also found to be the case, the sensation of crepitus being communicated to the hand which grasped the calf as well as to that which held the heel; he suffered intense pain during these movements. There was very great swelling and extravasation of blood, extending as high as the knee; when this had subsided sufficiently to allow of further examination, crepitus was no longer obtained by irritating the muscles of the leg, although it was produced by all the other manipulations before mentioned. The os calcis appeared to be broken about the junction of the posterior two thirds with its anterior third, and the fracture seemed to pass from above obliquely downwards and forwards. All movement was prevented by paste-board splints; but it was not until November 5th that he was able to walk at all. On the 21st of November he was discharged.

About a month afterwards he returned to the hospital, and on removing the splints, firm union was found to have taken place, with a great deposit of callus below and in front of the malleoli, which somewhat interfered with the movements of the joint, but quite confirmed the diagnosis of the fracture previously given.

Fracture of the os calcis appears to be regarded as a very rare accident by English writers, and is treated of very briefly by them; but Malgaigne, who has devoted much attention to, and published a memoir on, this subject, speaks of it as much more common than is usually imagined; and he cites several instances which occurred in the practice of others, as well as in his own.

In his treatise on fractures and luxations, he says, that "the fracture is always situated behind the astragalus;"

and Sanson, in his article on Fractures, in the 'Dictionnaire de Médecine et Chirurgie pratiques,' states, that "up to the present time it is only behind its articulation with the astragalus that we have found it fractured." But this instance would seem to disprove those statements, for the callus deposited shows that the fracture was situated in front of the larger articulating facet for the astragalus.

Portscript.—October. Since writing this case, I have found an instance of fracture of the anterior third of this bone, recorded in the 'London Journal of Medicine,' for January, 1851, as having occurred in the practice of Dr. Uhde, of Brunswick. Two other cases of fracture of this bone have also since been admitted into St. Mary's Hospital; the one on July 1st, in which the posterior and upper extremity was separated from the rest of the bone: caused by jumping from a height of eighteen feet, and alighting upon the heel. The other, on August 6th, in which the bone was broken at the junction of the posterior third with the anterior two thirds: caused by a fall from a window of some height, the heel first coming into contact with the ground.

TWO CASES
OF EXTENSIVE
ABSORPTION OF THE BONES OF THE HEAD
FOLLOWED, IN ONE OF THEM, BY
HERNIA CEREBRI.

BY
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Received May 26th.—Read May 27th, 1856.

CASE 1.—G. C—, æt. 24, was admitted into St. George's Hospital, under the care of Dr. Nairne, March 6th, 1850, with disease of the right lung, which commenced above a year before with severe inflammation, for which he was under treatment eight months. His present attack began, five weeks before his admission, with hæmoptysis, succeeded by sanguineous and muco-purulent sputa, and other signs of tubercles of the right lung.

Three days before his admission he felt pain on the right side of the head, soon followed by a swelling, without his having suffered in any way beforehand from any affection of this region. For this I was requested by Dr. Nairne to see him, and on the 15th, about twelve days after the occurrence of pain, I opened an abscess beneath the pericranium covering the lower part of the parietal bone. In

the early part of April, I opened a second abscess, and on the 12th, a third, so that a considerable part of the side of the cranium was now denuded of covering by the extension of the inflammation towards the occiput and forehead, the matter being unhealthy and scrofulous with flakes of lymph in it.

On the 19th, some purulent discharge took place from the right ear, which had not been affected at any previous time, and the disease was extending towards the vertex, where another incision was necessary.

Notwithstanding free discharge from these incisions, the disease, by the 29th, had extended across the vertex to the left side, where a fresh collection of pus required to be evacuated; nine or ten incisions being altogether required in different parts, while the pericranium still continued to adhere in some parts between them. The strength of the patient gradually declined, and sometimes in the last few days he seemed scarcely conscious,—from exhaustion rather than from any local affection of the brain,—and he sunk on May 18th, about ten or eleven weeks from the first sensation of pain in the head.

On examination, the scalp over the crown and right side of the head was found thickened and infiltrated with serum, and the pericranium was separated from the bones over almost the whole of these parts, except towards the anterior part of the frontal bone, and it was thickened and presented an appearance somewhat like brawn.

The entire parietal bone, the right half of the occipital, the squamous and mastoid portions of the temporal bone, and a considerable part of the frontal, together with a part of the greater wing of the sphenoid entering into the right zygomatic fossa, were affected on both surfaces with caries. Very delicate and thin layers of new bone, of a whitish colour, were deposited on the internal surface of the frontal and occipital bones, but only to a slight extent. The interior of the mastoid cells and the cavity of the tympanum were filled with purulent fluid, and their lining membrane was thickened. The small bones of the ear were entire; but

the membrana tympani was perforated. Corresponding to these affected parts of the cranial bones both the pericranium and dura mater were found altered, rough, and thickened, and detached from the bones, quantities of thin, flaky, purulent fluid intervening. The pericranium and dura mater were both thickened; but the smooth serous surface of the latter was unaffected. The superior longitudinal and right lateral sinuses, as far as the petrosal, were obliterated by fibrinous coagula. The brain was healthy.

Where least affected by caries the bones presented a worm-eaten appearance; but towards the front of the right side of the head large openings existed, where the bone had been entirely absorbed, around which other portions were very thin, though not yet entirely absorbed. No part whatever had died.

The cavity of the right pleura was completely obliterated by old adhesions, and the lung consolidated, with cavities towards the apex.

The kidneys were congested and the capsules adherent; and the liver was large and soft.

There is no doubt that the disease, in this consumptive patient, was scrofulous caries of the bones of the head; but, although the disease itself is not uncommon, this particular case may deserve attention on several grounds.

Scrofulous disease of the cranium usually occurs in one or more distinct tubercles, or deposits of strumous substance between the bone and the pericranium, or between the bone and the dura mater, which advance slowly towards suppuration, and, when situated externally, constitute strumous nodes of a round or oval shape, the periosteal covering shading gradually off into the undetached membrane. In a lad admitted for diseased hip into St. George's Hospital, cerebral excitement ending in coma, caused death in a few days after pain in the head was first complained of, and the early stage of this form of the disease is very well seen in the preparation on the table. In this case six or seven tubercles with roughness and excavations in the surface of the bone existed on one or other side of the cranium, and one in the sella

turcica, while the other parts of the bones were quite healthy, the immediate cause of death being some scrofulous tubercles in the brain.

Sometimes, however, after injury, and perhaps when a strumous state of the system is less strongly developed, there is general inflammation of the cranium over almost its whole extent, both surfaces being worm-eaten, as it were, with numerous depressions, filled with gelatinous fluid of various depths, or even entirely perforating the bone by numerous small openings. But even in these cases, as far as I have seen, if suppuration take place it is generally in separate small abscesses, like soft nodes, not numerous or over a great extent of surface at any one time, and only forming small perforations through the skull.

I have indeed seen the pericranium in children occasionally separated from the bone to a great extent; but it was in the form of acute inflammation without caries, or scrofulous deposit, and when opened by a small puncture, if pressure be employed, the pericranium may again unite with the bone without any exfoliation, or with the loss of a very small portion in the seat of the puncture, even when the abscess covers above half the head.

In the cases before described, the chronic nature of the disease is generally well marked, as successive portions assume from time to time a more advanced stage of suppuration and caries; and when a similar worm-eaten appearance of the skull is occasioned by the effects of syphilis and mercury, the disease is also found to be of a chronic kind, with the additional fact of extensive necrosis in many of the worst cases.

In this case, on the contrary, the disease commenced in one part of the side of the head, the first abscess being opened as early as the twelfth day after pain was first felt. From this part the inflammation spread with unexampled rapidity in all directions, though matter formed more rapidly in some parts than in others, so as to require separate openings; the whole duration of the case being little more than ten weeks, in which time more than half the cranium



became affected on both surfaces with the immense destruction observed in the preparation, and already described, instead of the small perforations usually observed.

It will have been noticed that suppuration existed in the mastoid cells and cavity of the tympanum and outer ear; but the inflammation clearly spread to the ear from the side of the head, where it commenced, and therefore the several parts of the ear were perfect in structure, and the patient had been quite free from previous affection of this organ.

The second case was in every respect, except the great loss of bone by absorption, different from the former, and differed in many respects from any other case I have seen.

CASE 2.—C. L—, æt. 36, was admitted into St. George's Hospital, under Mr. Keate, on the 2d of February, 1832, with a pulsating tumour, nearly five inches in diameter, on the upper and posterior part of the right side of the head; it was soft and elastic, with a well-defined boundary, and the pulsation could be felt and seen over its whole extent; in its centre a round fungus projected through an opening in the scalp, of the size of a small walnut, in which pulsation was also evident, and the apex of the fungus looked like layers of coagulated blood. The tumour was not tender to the touch, nor was there any apparent change over the adjoining bone. On the opposite side of the head, in the superior part of the left parietal bone, was a circular depression, about three inches in circumference, which was soft and elastic, and pulsated strongly, the edges of the bone not being abrupt or well marked round the aperture, and pulsation could also be felt in another smaller depression near this large one.

He complained of occasional pain in his head, which became very severe if he took any kind of stimulant; he was aware of some confusion in his ideas occasionally, and,

if he thought much, a sensation of giddiness came over him; on lying down, or lifting his head from the pillow, he felt the tumour pulsate strongly; and the least coughing, or straining, or stooping forwards, caused great pain in the tumour, which sometimes bled slightly. At night his eyes were dim, and *muscæ volitantes* passed over them. He had perfect use of his limbs, and passed his water naturally; his countenance was yellowish, and rather flushed.

He had lived freely, and had been in the East and West Indies as a sailor and gentleman's servant, but had enjoyed good health, except a tendency to rheumatism; twelve or fourteen years ago he had twice been salivated for chancre, but had not suffered since that time.

It appeared that two years and a half before, while asleep on the grass in the sun, he suddenly felt acute pain in the head, which remained more or less for several days, incapacitating him for work. Some time afterwards, the exact period being unknown, he first perceived a small depression in the situation of the tumour, of sufficient size to admit the end of his little finger. This depression went on increasing for six months, making greater progress when he suffered from cold, or when he had drank freely, the pain being at those times very severe; and at last a tumour formed instead of the former depression. Eighteen months ago, and a year after the first seizure, he was laid up on account of the severity of the pain, and the increase of the tumour, and at this time his attention was drawn by his surgeon to its pulsation, which he had not himself noticed. Poultices were applied, and the tumour was punctured with a lancet, but only a little blood escaped. Attempts were made to induce suppuration, but the puncture healed, and he went about his usual avocations, suffering no great pain. A month afterwards it was again punctured, but with the same result, so that it may be presumed that the tumour was supposed to have been abscess.

From this time nothing more was done, the tumour not increasing in size, and only giving him occasional pain, and

not materially affecting his health till eight weeks ago, at which time, while walking in the street, he suddenly felt a sharp pain in the head, with giddiness and partial insensibility, which caused him to fall against the wall. He was supported home, and bled in both arms, and had leeches applied, and the tumour was punctured a third time, but again, as he believed, only blood escaped. He was only laid up for four or five days, and then went about again; but from that time the present fungus began to protrude through the opening, and has gradually increased. He has also some enlargement, like periosteal thickening, around the lower part of the femur, which is rather uneven, but not tender nor painful.

Some difference of opinion existed on his admission, on the 2d of February, as to the nature of this fungous tumour, whether it originated in the bone or dura mater, but its cerebral origin was, I believe, unsuspected; though at the present time it would probably be readily detected by microscopical examination. With such a fungus, however, in one part, and an aperture in the skull at another, it is not surprising that fungous tumour of the dura mater, or of the *diplôe*, should be suspected, rather than so rare a case as that of hernia of the brain from disease of the bone.

On the 8th, a ligature was passed, not very tightly, round the projecting fungus, which sloughed off on the 16th; and on this day Mr. Keate passed a needle, armed with a double ligature, across the base of the tumour, and tied each half separately, without the production of any bleeding.

On the 22d, except occasional headaches, nothing particular had occurred, and two or three portions of the tumour having sloughed off, and other parts being dead, the tumour seemed much less in size.

On the 25th, he was somewhat feverish, with shooting and throbbing pain in the head; the ligatures had separated with some more sloughs, but the tumour was again growing, with increased pulsation. This was partly relieved by bleeding.

On the 27th, there was some numbness of the right leg and left arm, and some dimness of sight, especially in the right eye, with increased giddiness on moving, accompanied by some fever and disposition to sickness.

On the 29th, a large part of the fungus came away, of a brownish colour, looking like fibrous coagulum, with soft blood-vessels, containing coagulated blood, and in parts the sloughs looked like medullary tumour around such blood-vessels. He was less feverish and ill.

On March 6th, though feeling better the last few days, he was languid and drowsy, and had some partial paralysis of the left side of the face, and the movements of the left arm were much impaired, with numbness of this arm, and, to a less extent, of the left leg also.

On the 8th, this arm was entirely paralysed, and the tumour was larger and more sloughy.

On the 12th, the whole of the left side was paralysed, and the right leg partially so; the sphincters were relaxed, and the evacuations passed unconsciously, and he had some difficulty in swallowing. The tumour was increasing, although portions sloughed off.

On the 16th, he became comatose, and died on the 20th of March.

The nature of this pulsating tumour was at once rendered plain by examination, and may be clearly seen in three preparations on the table, preserved in the Museum of St. George's Hospital.

The greater part of the calvaria has been dried, and it will be seen that towards the anterior part the bones are, to a great extent, very much thickened, being in some places half an inch thick; their tissue is firm and dense, like ivory, the *diplœ* being obliterated. Towards the posterior part, they are very thin, and, in many places, present large apertures, where, during life, the pulsations of the brain could be easily felt. The external surface of these bones is, for the greater part, rough and mamillated,



and the corresponding internal surface more porous than natural.

In another preparation, the remaining portion of the skull-cap has been preserved in spirit, in order to show the opening in the scalp through which the protrusion of the brain had taken place, and in it will be seen the complete adhesion of the dura mater to the integuments, while portions of the arachnoid and pia mater are observed hanging from the inner surface of the dura mater, to which they adhered, round the opening.

It will be observed, in a third preparation, that the fungus was not of large size at the time of death, and that an opening in its centre leads into an abscess of considerable size in the right hemisphere, almost as deep as the lateral ventricle, the purulent contents of the abscess having been discharged through the opening during life. The cerebral substance around the abscess and fungus, was vascular, and altered in colour, but was not otherwise diseased.

Every surgeon is familiar with the protrusion of the brain, which follows many cases of injury of the head, of recent occurrence, in which the removal of the bone leaves the dura mater exposed; but hernia cerebri from disease of the bones of the cranium is comparatively a very rare occurrence. The reason of this is obviously the chronic inflammation of this membrane, by which it is strengthened, so as to bear the loss of the osseous covering in cases of ulceration and necrosis, before the aperture is formed in the bone. Such thickening of dura mater also slowly follows injuries, when the patient survives the removal of portions of the cranium. In this case, on the contrary, if the edges of the opening are examined, it will be seen that the pericranium and the dura mater are both in a perfectly normal condition, even close to the irregular margins of the bone; and therefore the pressure of the dura mater against these rough edges appears to have been as injurious to the membrane as in cases of recent injury.

I have not myself seen *hernia cerebri*, even from extensive exposure of the dura mater after necrosis, and I am inclined to believe it must be still more unusual, when the integuments are entire, as in this case.

That the disease commenced in the bone, and not in the brain, must be evident from the depressions which preceded the protrusion, from the extensive disease of the calvaria in other parts, and from the fact that below the smaller apertures, in which the pulsation of the brain was seen without protrusion, the dura mater and the brain were quite unchanged. Here, therefore, the increasing size of the aperture in the bone might have been followed by the same results, if the patient had survived much longer, and the edges had become as irregular as those of the opening through which the hernia had taken place.

The exact nature of the disease in the bone may admit of some doubt, as it is, I believe, very unusual for the cranial bones in chronic inflammation to be seen with marks of so much healthy action as in this case. The ivory, solid thickening of some parts of the bone, and dense nature of the mamillated portions even among the ulcerated parts, where the bone had been thinned by absorption, are remarkably different from the soft worm-eaten appearance of the more common caries of the first case. It may, perhaps, be thought, from the patient having been subject to rheumatism, from the enlarged condition of the femur at its lower part, and from the commencement of the disease, while the man was lying on the grass, that the inflammation of the bone was of a rheumatic character; but if this conjecture be correct, such extensive absorption of the bone is at least very unusual; though an analogous ivory deposit on the articular ends of the femur and tibia, with depressions from absorption, are certainly sometimes seen in long-continued rheumatic inflammation of the knee joint.

There is nothing in the history of the case, or in the morbid appearances, which at all point to a syphilitic origin.

Supposing, however, that the disease was inflammatory,

whether rheumatic or otherwise, it seems impossible to give any explanation of the extensive absorption at one part, while other parts were immensely increased in density.

The opening in the bone having been formed by absorption, was succeeded, after an interval of at least some months, by a swelling of much greater diameter than the orifice in the bone, as if the dura mater had given way long before the small fungus protruded through the bone, and the cerebral matter had spread out between the bone and the integuments; but it may admit of doubt whether the brain would have forced its way through the scalp, had not repeated punctures been made by the surgeon's lancet—the last of these punctures having never healed, although the former ones had done so; and the fungus having soon followed the final puncture.

of production, which is a consequence of the fact that the production process is not a simple one, but a complex one, involving a large number of different activities and a large number of different inputs.

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ON TWO CASES
OF
MALFORMATION OF THE HEART.

BY
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COMMUNICATED BY
SPENCER SMITH, Hon. Sec.

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THE two cases of malformation of the heart described in this memoir are of extreme interest ; the first, as an instance of anticipation of the adult condition of the heart during foetal life ; and the second, as an instance of that rare and opposite condition, in which the three great elementary cavities of the heart have undergone hardly any fission at all.

CASE 1.—On January 5th, 1856, I delivered a woman of a female child, which gave hardly any indications of vitality for some minutes after birth. It ultimately respired pretty freely, but soon became very blue, and about an hour after birth relapsed into its original feebleness. The pulse became weaker, slower, and intermittent. On applying my ear to the infant's chest, I found that the natural cardiac sounds were replaced by two loud blowing murmurs ; the rhythm of these murmurs corresponded to the rhythm of the natural sounds—thus there was first *bruit*, second *bruit*, and pause—and the number of rhythms per minute was but little below the standard of a healthy new-born child. Blueness

of the surface increased, difficulty of breathing supervened about two hours after birth, and augmented rapidly, and four hours and a half after birth, having been convulsed two or three times, it died.

On examining the body of the infant three days after its decease, the following abnormalities of the thoracic viscera were discovered. As regards the heart—the right auricle was larger than natural, and its parietes were very thin. The foramen ovale was closed, or in other words the septum of the auricles was totally imperforate, there being nothing more than an oval depression in the usual site of the foramen. The tricuspid valve was incompetent; and formed rather a festooned imperfect diaphragm between the auricle and ventricle, than a valve. The right ventricle gave origin to two vessels; one of these immediately divided into a right and left pulmonary artery, it neither communicated with nor was attached to the aorta, and was of less calibre than usual; the second vessel was the aorta, which arose in part from this cavity, and in part from the left ventricle. The common origin of the aorta from both ventricles was the result of an imperfect septum ventriculorum, the deficiency occurring at the upper part of the septum. The left ventricle was small; the mitral valve was perfect; the left ventricle communicated with the right ventricle at its upper part, as before described, and gave partial origin to the aorta; the semilunar valves were perfect. The lungs were very imperfectly inflated, and were situated at the back of the chest, purple, and gorged with blood. The rest of the body was apparently well formed, but rather smaller than usual.

The course of the circulation during intra-uterine life would appear to have been as follows: all the blood returned by the *venæ cavæ* into the right auricle, except that which regurgitated, was propelled through the right ventricle into the aorta. A small proportion only of blood passed into the pulmonary artery; the left ventricle was only concerned in returning to the general circulation the small quantity of blood which had passed through the lungs. Thus before

birth there was regurgitation of blood through the tricuspid valve, and admixture of venous and arterial blood in the aorta at its very commencement. The physiological result was that the head and upper extremities, instead of receiving better oxygenated blood than the trunk and lower extremities, received blood of similar quality. As soon as birth took place, and the left instead of the right side of the heart became the arterial side, the faulty development, and the narrowing of the pulmonary artery became productive of fatal consequences. The errors of circulation after birth were three—

- I. Regurgitation from tricuspid incompetency.
- II. Admixture in the aorta of venous and arterial blood, from imperfection of the septum ventriculorum.
- III. Deficient supply of blood to the lungs, from narrowing of the pulmonary artery.

If it were not for one circumstance, I should not have thought this case worthy of particular notice. It is the type, but for this exceptional addition to its abnormalities, of a large class of malformations of the heart. Partial deficiency of the septum ventriculorum, with narrowing of the pulmonary artery, is, perhaps, the commonest cardiac defect, of a congenital origin, known; with, perhaps, the exception of patency of the foramen ovale. But the fact that the septum of the auricles was totally imperforate, and that there was for a considerable time during foetal life no communication between the right and left auricles, rescues the case from the category of familiar irregularities of development. Malformations of the heart are in general "per defectum;" the present one is manifestly "per excessum." Imperforation of the cavities of the heart is said to be extremely rare. M. Geoffroy St. Hilaire states, in his work 'On Anomalies of the Organization,' that an instance of it had never come under his own observation. Whether this statement refers to an imperforate condition of the septum of the auricles at birth, I cannot say, but

from an attentive perusal of that section which treats of abnormalities of the vascular system, I have been unable to discover that that eminent teratologist was acquainted with the condition in question. Since the time of St. Hilaire, however, a case has been recorded by Mr. Ebenezer Smith, in the first volume of the 'Pathological Transactions.' The history of this case is very similar to mine, but the child lived twenty-one hours. The points of difference are two; in the first place, it appears that the closure depended simply upon adhesion of the valve of the foramen to its margins, whereas in the case I have related, the septum of the auricles was fleshy throughout; and secondly, in Mr. Smith's case, the pulmonary artery was wider than natural, while it was narrower in mine. Dr. Peacock informs me that another case of premature closure of the foramen ovale is recorded by Vieussens, but I am unable to refer to it more particularly, having failed to discover where it is related.

CASE 2.—On May 12th, 1856, I delivered a woman of an apparently healthy and robust male infant. On the 18th, I discontinued attendance, as both mother and child appeared to be perfectly well. Early in the morning of the 20th, a messenger was sent to say that the child had just died. The account given was that on the previous afternoon the infant began to pant, that the difficulty of breathing increased, and the skin became discoloured, that during the night the child was convulsed, and that it died at 7 o'clock a.m. The cause of death not being apparent, I demanded a post-mortem examination of the body.


The heart presented the following singular series of defects; The right auricle was large and more fleshy than usual. The foramen ovale was of greater area than in the normal condition. The valve of the foramen was unusually large and membranous, and was attached posteriorly to a fleshy column, which arose from the upper and posterior part of the auricle, and inserted itself into the ring at the base of the ventricular portion of the heart, rather to the left. This

fleshy column constituted, together with the valve of the foramen ovale, all that existed of an inter-auricular septum. The left auricle was very small, and, in fact, a mere diverticulum from the right auricle; it received *two* pulmonary veins, one from each lung, and communicated with no other cavity than the right auricle. The ventricular portion of the heart was single, and did not present the slightest vestige of a septum. The valve between the right auricle and the general ventricle, consisted of two parts. The largest of these was anterior, and to the left; it was attached, in the usual manner, by cordæ tendineæ to two well-defined papillary muscles of considerable thickness; the largest of these muscles arose from the anterior surface of the cavity of the ventricle, about one third of the length of the whole cavity from the apex of the ventricle. The smaller papillary muscle was attached by its base to the extreme right of the ventricle, and it was not so long as the other muscle; its cordæ tendineæ were not all inserted into the larger or anterior segment of the valve, some went to the posterior division. The posterior segment was situated more to the right than the former one; it was not so free, inasmuch as the tendinous cords attached to it were very short, and arose principally from columnæ carneæ on the posterior surface of the ventricle; it was neither so wide at its base nor so deep as the anterior division of the valve; it had not the distinct apex of the other portion, and its free border was wavy and irregular. A single vessel arose from the general ventricular cavity; it was much larger than either the aorta or the pulmonary artery taken alone, and was more decidedly bulbous at its origin than the normal aorta. The branches from the main trunk were as follows: first, two vessels arising from the posterior aspect of the ascending part of the aorta, these were a right and left pulmonary artery, and their aortic embouchures were close together. Then a third vessel arose from the summit of the aortic arch; this divided into the innominata, left common carotid, and left subclavian arteries. The aorta then continued its course downwards, as usual. No coronary arteries could be discovered,

until attention was directed to a vessel which coursed downwards, towards the base of the heart, from the innominate artery; at first this vessel appeared to terminate in a cul de sac, and I was at a loss to determine what structure it represented; subsequently, after more careful examination, it proved to be a common coronary artery.

The heart and great vessels were distended with black blood. The lungs were imperfectly expanded, so much so that their bulk was certainly not equal to more than one third of the usual dimensions; they were slightly crepitant throughout, more so at the apices than the bases; they were liver-coloured, and situated quite at the back of the chest, and did not overlap the heart, or displace the abdominal viscera sensibly from their foetal position. The most striking phenomenon in connection with the pulmonary organs was, that scattered over the anterior aspect of the superior lobes, there were small rosy coloured elevations, about the size of a millet-seed; each of these elevations appeared to consist of isolated portions of expanded pulmonary tissue. On inflating the lungs by blowing down the trachea, these patches disappeared, and the lungs then presented an homogeneous surface of the colour of the expanded lung in children newly born.

The course of the circulation before and after birth would appear to have been the same. In consequence of the non-communication of the left auricle with the ventricular cavity, the right auricle was the recipient of the aerated blood after as well as before birth; and all the venous blood in the body, whether belonging to the pulmonary or systemic circulation, found its way into that cavity. From the right auricle, the blood passed into the general ventricle, and was then propelled into the general aorta; from this vessel came off two pulmonary arteries, which were wholly inadequate to the purpose of passing a sufficient quantity of blood to the lungs. The nature of the error of circulation after birth would appear to have been this: that instead of the whole mass of the venous blood being passed through the lungs before being again sent into the systemic capillaries, only



about one third or one fourth was propelled into the aerating organ, and that even this fractional part of the blood, instead of finding its way into the systemic capillaries, was again partially diverted into the pulmonary circulation. Assuming, for purposes of illustration, that one fourth of the blood projected from the ventricular cavity found its way into the pulmonary circulation, and that that blood, on its return from the lungs, became intimately mixed with the blood in the right auricle, it would follow, that one fourth of the decarbonized blood, or one sixteenth of the whole mass, re-entered the pulmonary circulation, and that three fourths of the aerated blood, or three sixteenths of all the blood propelled into the aorta was aerated blood destined for the service of the economy at large.

It is perhaps hardly possible to conceive a heart more radically malformed, which should even for a short time subserve the necessities of the economy; the veins, the cavities, the valves, and the arteries, all deviate widely from the normal condition. There are certainly very few instances recorded of such extreme deformity, and I do not know of one exactly similar. Subjoined is a sketch of some of the most analogous cases hitherto published, and other cases are referred to merely.

A. In the first volume of the 'Pathological Transactions,' there is a case related by Mr. Forster, more like mine than any I have met with. There was one large auricle with the same number of veins opening into it, viz., five; the valve appeared to have been similar; the single ventricle gave off a single vessel, and the arrangement of the branches from this aorta was like that in the case described above, except that the coronary artery came off from the concavity of the arch of the aorta, instead of from the innominate, and it subdivided before reaching the heart. There was no rudimentary left auricle in Mr. Forster's case. The child lived seventy-eight hours, and died cold and livid during an attack of dyspnoea.

B. Dr. Ramsbotham reports a case on the same page of the 'Pathological Transactions' as Mr. Forster's, very similar

to both the preceding ones. There was only one auricle and one ventricle. A single vessel arose from the ventricle, and from this a branch was given off which subdivided into two pulmonary arteries. The child lived ten days, and was completely cyanotic, but neither respiration, temperature, nor muscular action appeared to be affected. The case is figured and described in the ninety-ninth vol. of the 'Philosophical Transactions.'

c. A case is described at page 49 of the first vol. of the 'Pathological Transactions,' by Mr. Crisp, in which there was a single auricle and a single ventricle. A single vessel arose from the ventricle, which divided near its origin into branches, the destination of which is only surmised. There was a rudimentary vessel also behind the aorta, and another artery, the origin and destination of which does not appear to have been determined.

d. A case is related by Martin Saint Ange, in the third number of the 'Bullet. de la Société Anatomique' for 1826, of a child which died six weeks after birth, having been subject to convulsions and vomitings. There was general transposition of the viscera. The heart consisted of one auricle and one ventricle; but of the state of the arteries I cannot find a description. The colour of the skin was livid, the child was habitually somnolent, and the temperature of the back and lower extremities was lower than that of the rest of the body.

e. Dr. Farre relates a case, in his 'Essay on the Heart,' which very closely resembles Mr. Forster's case and mine. The child survived its birth 79 hours. The respiration became hurried, the diaphragm laboured excessively, the skin became cold, and the face somewhat livid, but not blue. Torpor ultimately came on, and the respiration became more and more feeble until death occurred. There was a single auricle, a single ventricle, and a single artery from the ventricle. The single artery behaved as in the case I have related, except that the coronary vessels were differently arranged. There was not either any rudimentary left auricle.

F. Jackson relates the case of a child which died at the age of three years, in which the respiration was very irregular; but there was no blueness of the skin. The inter-auricular orifice was found patent, and there was a large opening in the inter-ventricular septum. The arteries were also abnormal. This case is a minor instance of the same developmental defect, which in the preceding cases resulted in hearts practically single.

G. There is a case described by Professor Owen, in vol. 2, p. 664, of the 'Lancet' for 1848, which is very similar to mine.

H. Dr. Hale's case, in the 4th vol. of the 'Pathological Transactions' is another intermediate case of malformation. The auricular portion of the heart was double, the ventricular single, and there were two main trunks arising from the ventricular cavity. The child lived nineteen weeks, subject to dyspnoea and vomitings; it was found dead in bed one morning. Dr. Hale refers to analogous cases by Kreysig, Tiedemann, and Hufeland. His own case does not appear to have been cyanotic. Numerous other cases of malformation of the heart are described by Fleischman, Chemineau, Otto, Dr. Cheevers, and others; but none, I believe, present a more complete arrest of development than the cases reported by Mr. Forster and myself.

In speaking of the case I have related as an instance of arrest of development, I refer, of course, to the well-known fact that, before about the ninth week of embryonic life, the rudimentary heart consists merely of three dilatations of the main vascular trunk: the auricular sinus, the ventricular sinus, and the bulbus arteriosus. At the period mentioned, ridges appear in three sinuses which meet in the centre, and ultimately divide each cavity into two.

Reference to the cases I have quoted and referred to will justify, I think, the conclusion that cyanosis is the constant attendant of no single species of malformation of the heart. Dr. Reeder advanced an opinion that, in cases where there was patency of the foramen ovale or an hiatus in the inter-

ventricular septum, cyanosis or other inconvenience does not necessarily occur so long as no obstruction is offered to the course of the blood in the lungs, or elsewhere, and for the following reason, viz., that the right and left sides of the heart being nearly equal in size, acting simultaneously and with nearly equal force, it does not at all follow that any great amount of admixture of blood shall take place simply because the cavities communicate; but as soon as any obstacle is offered to the exit of the blood either from the right or left sides of the heart, the balance is destroyed, and admixture then takes place. This explanation seems applicable enough to a limited number of cases; but it will not account for all. The following general conclusions with regard to cyanosis appear to be safe:

1. That it may be connected with abnormalities of the great vessels only.

2. That it is not so necessarily connected with abnormalities of the orifices of the heart as always to be present when they are.

3. That there may not only be patency of the foramen ovale, but defect in the interventricular septum also, and yet no blue disease.

4. That it may be the result of veritable pathological changes, such as adhesions of the lungs to the pleuræ and pericardium (*vide* Marc, Tartra, and Gilbert, in the 'Bullet. des Sc. Med. '); and

5. That if any one condition be more inseparably connected with cyanosis than another, it is narrowing of the pulmonary artery or such abnormality as diminishes the supply of blood to the lungs, especially if such abnormality be of congenital origin.

EXPLANATIONS OF THE FIGURES.

FIG. 1.

- A, A, A, or a, a, a.* The general aorta.
- b.* The aortic embouchures of the right and left pulmonary arteries.
- c, c.* The origin and trunk of the brachio-cephalic vessel.
- d.* The left subclavian artery.
- e.* The left common carotid artery.
- f.* The innominate artery, which afterwards divided into the right common carotid and right subclavian arteries.
- g.* The common coronary artery, coursing downwards from the innominate artery to the base of the heart.
- h.* A probe passing through the left auricle and the imperfect septum aricularum into the right auricle, and thence into the cavity of the general ventricle, where its other extremity may be seen.

The simular valves may be seen to be perfect, and the anterior segment of the auriculo-ventricular valve is conspicuous in the general ventricular cavity. The aorta is divided longitudinally.

FIG. 2.

- a.* The left or anterior segment of the auriculo-ventricular valve.
- a'.* The papillary muscle of segment *a*.
- b.* The right or posterior portion of the same valve.
- b'.* The papillary muscle of *b*.
- c.* The fleshy column, which, with the valve of the foramen ovale and another small column (*e*), constitutes the rudimentary septum auricularum.
- d.* The valve of the foramen ovale.
- e.* A small fleshy column, which defines the position of the foramen ovale.
- f.* A probe passed behind the valve, &c., into the left auricle, and thence into the right auricle again.



ON
ENCEPHALOCELE;
BEING THE
HISTORY OF A CASE,
WITH
A TABULAR ANALYSIS OF SEVENTY-FIVE CASES.

BY
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COMMUNICATED BY
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CONGENITAL hernia of the brain is a rare malformation; for the subjects of it to live any length of time is a matter of still greater rarity. In the following instance of the disease life was considerably prolonged, and it is chiefly on account of this peculiarity that I have ventured to submit the case to the Society.

Mrs. G— was delivered, on the 15th of November, 1855, of a female child, which bore at its birth a tumour at the back of the head. The labour was natural.

The child was two months and a half old when I first saw it. Concealing, and apparently originating from, the occiput and nucha, was a somewhat conical tumour, nearly as large as the child's head, measuring five inches and a

quarter in length, and three inches and a half from side to side. The tumour was bounded by a thin, glossy membrane—stretched skin of the head and neck—upon which coursed numerous varicose veins, and which had hairs on its upper surface similar to those of the head. (See the figure.) So freely did the tumour fluctuate under the fingers, that it felt like a thin bladder filled with water; and when one half was shaded by the hand, the other half transmitted even the



daylight in a marked degree. There was no pulsation perceptible. The child had a very receding forehead, and a vacant idiotic stare; was to all appearance blind, but was startled by any sudden noise. The limbs were not paralysed, on the contrary, the flexor muscles of the right hand were in a state of tonic contraction; the fingers of this hand were constantly kept clenched and turned inwards, and any attempt to unclench the hand made the child cry. The body generally was very thin and puny. It had been wasting ever since birth (although the child took a good deal of food), whilst the tumour had increased rapidly in size. Shortly before death, contraction of the fingers of the left

hand occurred. The mother, who was a healthy looking young woman, thirty-one years of age, had previously given birth to five hearty children. She found the child dead by her side on awaking, the morning of the 8th of April, 1856; so that it had lived altogether about five months.

Autopsy ten hours after death.—The tumour having been cut into, about half a pint of clear colourless fluid escaped; this fluid was alkaline and albuminous. When the tumour was laid open, it was seen to contain a portion of the brain, and the entire cerebellum; these were continuous with the intracranial portion of the encephalon. The cavity of the tumour was lined by the dura mater and the arachnoid, between which and the portion of contained cerebrum passed several bands of old adhesions. The intracranial portion of the brain offered no anomaly of conformation; but the longitudinal fissure was prolonged back through the extracranial portion, which was thus divided into two halves; further dissection proved these to be two large cavities (three inches and a half long, by two inches and a half transversely), each containing the choroid plexus, and each continuous with the corresponding lateral ventricle. The cerebellum was greatly atrophied, especially its left half, the margin of which did not exceed one eighth of an inch in thickness. The cerebral substance and the cranial nerves appeared normal.

After the brain was removed, a hole was perceived in the occipital bone; this hole was oval in form, continuous with the foramen magnum, and measured (including this latter) two inches and a quarter from before backwards, and one inch and a quarter across. The margins of the aperture corresponded with the lateral sinuses. The basilar and condyloid portions of the occipital bone were of their usual proportions, and the articulation of the condyles was perfect; but immediately above the commencement of the spinal canal was an expanded osseous cavity, on each side of which was the foramen lacerum jugulare, with its vein and nerves.

I am indebted to Mr. Jakins, of Osnaburgh Street, for the opportunity of observing this case.

A TABULAR ARRANGEMENT OF SEVENTY-

I. *Of those occurring in*

No.	Sex.	Age at Death.	Size.	Part of Brain protruded.	State of Cerebrum.	State of Cerebellum.	State of Spinal Cord.
1	M.	47 days (operated on).	Of two walnuts.	—	Softened.	Gangrened.	—
2	M.	95 days.	Of a walnut.	Cerebellum.	Effusion into ventricles.	—	Spina bifida.
3	M.	2 days (operated on).	6 in. \times 5 in.	Posterior lobes of cerebrum and cerebellum.	Effusion into ventricles.	—	—
4	—	Still-born.	6 in. wide.	Posterior lobes of cerebrum.	—	—	—
5	M.	26 days.	4½ in. across.	—	Atrophied; effusion into ventricles.	—	—
6	—	Fœtal.	Of the child's head.	—	—	—	—
7	—	— (Operated on.)	Of the child's head.	—	Sound.	—	—
8	M.	Some days (operated on).	3½ in. long.	—	—	—	—
9	—	34 days.	—	Cerebellum.	Sound.	Too large.	Spina bifida.
10	—	6 weeks.	—	—	—	Soft.	—
11	—	Still-born.	Of the child's head.	Posterior lobes of cerebrum and cerebellum.	Atrophied.	—	Spina bifida.
12	—	Fœtal.	—	—	—	—	—
13	—	Still-born.	—	Whole of cerebrum.	Defective.	—	Spina bifida.

FIVE CASES OF ENCEPHALOCELE.

the Occipital Region.

State of Bones (at Hernia).	Other Malformations.	Reference to Author.
—	—	Salomon Reiselius; Ephemerid. Med. Physic. Acad. Nat. Cur., 1683, p. 272.
Hole, size of finger in occipital.	—	Lechel; <i>ibid.</i> for 1684.
Hole in proral part of occipital.	—	Corvinus; Haller's Disputat. Chirurg., t. ii, 1749.
—	—	Fried; Lieutaud's Hist. Anatom. medic., 1767.
Hole in occipital, near lambdoid angle.	—	Siebold; Collect. Observat. Medico-chirurg., 1769.
—	—	J. Gardner; Medical Commentaries, vol. v, p. 306, 1777.
—	—	Senac. Traité du Cœur, 2me éd., 1783.
Middle and right lateral parts of occipital perforated.	—	Thiebault, Desault's Journal de Chir., t. iii, p. 327, 1791-2.
Occipital, parietal, and part of temporal bones absent.	—	Penada, Ernia del Cerebro, 1793.
—	—	Ibid.
Hole in occipital continuous with foramen magnum.	Fusion of all the cervical vertebrae.	Van der Laar, Observat. chirurg., obstetric., anatomic., medic., 1794.
—	Umbilical hernia.	Sandifort, Museum Anatomic., pl. 126, 1793-1827.
Superior portion of occipital wanting.	Spinal canal open from neck to sacrum; union of three ribs; club-foot.	Hull, Memoirs of the Lit. and Phil. Society of Manchester, vol. v, p. 494, 1802.

No.	Sex.	Age at Death.	Size.	Part of Brain protruded.	State of Cerebrum.	State of Cerebellum.	State of Spinal Cord.
14	—	Still-born.	Very large.	—	Effusion into lateral ventricles.	—	—
15	—	30 days.	Of a fist.	Cerebellum.	—	—	—
16	F.	Still-born.	1½ in. wide.	Posterior lobes of cerebrum.	Atrophied.	Normal.	Spina bifida.
17	M.	Still-born.	—	—	Atrophied.	—	Spina bifida.
18	—	Fœtal.	Of the child's head.	Cerebellum.	—	Greatly extended.	—
19	—	Still-born.	—	—	—	Degenerated.	Spina bifida.
20	—	13 days.	—	—	Atrophied.	Small.	—
21	F.	17 days (operated on).	Larger than the child's head.	Posterior lobes of cerebrum.	Effusion into ventricles.	Normal.	—
22	F.	— (Operated on.)	—	—	Effusion into ventricles.	Atrophied.	—
23	M.	Still-born.	Size of head.	Posterior two thirds of cerebrum and cerebellum.	—	—	—
24	M.	Still-born.	—	Cerebellum.	Healthy.	—	—
25	F.	Still-born.	—	Posterior lobe of right hemisphere.	Atrophied.	—	—
26	—	Fœtal.	—	—	—	—	Spina bifida.
27	M.	—	Voluminous.	Greater part of brain.	Effusion into ventricles.	—	—
28	M.	—	Of a goose-egg.	Left posterior lobe of cerebrum, and left lobe of cerebellum.	Effusion into left ventricle.	—	—
29	—	9 days (operated on).	Of an orange.	Posterior lobes, and cerebellum.	Softened.	—	—
30	—	7 weeks.	Of a large orange.	—	—	—	—
31	—	Still-born.	—	—	—	—	—
32	—	44 days (operated on).	8½ in. round.	Posterior lobes of cerebrum.	Effusion into ventricles.	—	—

State of Bones (at Hernia).	Other Malformations.	Reference to Author.
Occipital perforated above foramen magnum.	—	Chaussier, Dict. des Sciences Médicales, t. xxxiv, p. 229, 1819.
—	—	Isenflamm, Anatomische Untersuchungen, 1822.
Hole, above occipital protuberance.	Cleft palate; club-foot; supernumerary fingers and toes.	Meckel, Deutsches Archiv, t. vii, 1822.
Hole, above occipital protuberance.	Cleft palate; club-foot; supernumerary fingers and toes.	Ibid.
—	Umbilical hernia of all abdominal viscera; left forearm absent; club-foot, &c.	Rathke, <i>ibid.</i>
—	—	Dugès, Ephem. médic. de Montpellier, t. ii, p. 289, 1826.
—	—	Cruveilhier; Anatom. Patholog., 1829.
Hernia came through posterior fontanelle.	—	Horner, American Journal of Medical Science, for 1829, vol. iv, p. 530.
—	—	Breschet, Archives génér. de Médecine, t. xxv, 1831.
—	Malposition of right hand; thumb of this hand wanting.	Ibid.
Occipital absent.	Absence of neck; diaphragmatic hernia.	Breschet, loc. cit.
Occipital perforated at lambdoid angle.	—	Ibid., t. xxvi, 1831.
Occipital perforated above foramen magnum.	—	Ibid.
Vast fissure of occipital, continuous with foramen magnum.	—	Roux, <i>ibid.</i>
Hole in occipital, continuous with foramen magnum.	—	Buettner, Dissert. inaug. Berol., 1832.
Hole in occipital continuous with foramen magnum.	Cranium very small; absence of fontanelles.	Robert Adams; Dublin Journal of Medical and Chemical Science, 1833, vol. ii, p. 338.
—	—	Ibid., p. 393.
—	—	Cloquet, Dict. de Médecine, vol. xii, 1835.
Hole in proral part of occipital.	—	Thierry, L'Expérience, for 1837.

No.	Sex.	Age at Death.	Size.	Part of Brain protruded.	State of Cerebrum.	State of Cerebellum.	State of Spinal Cord.
33	M.	Fœtal.	Large.	Posterior lobes of cerebrum and cerebellum.	Softened; right hemisphere small.	Very small.	—
34	F.	Still-born.	Large.	Posterior lobes of cerebrum and cerebellum.	Softened; effusion into ventricles.	Normal.	Spina bifida reaching to the sacrum.
35	F.	2 months.	Of a fist.	Posterior lobes of cerebrum.	Atrophied; effusion into ventricles.	Less than usual.	—
36	M.	Still-born.	Large.	Posterior lobes of cerebrum.	Softened; effusion into ventricles.	Very small.	Traversed by a large canal.
37	M.	Fœtal.	Large.	Posterior lobes of cerebrum.	Effusion into ventricles.	Very small.	Spina bifida reaching to pelvis.
38	M.	Fœtal.	Very large.	Posterior lobes of cerebrum.	Sound.	—	Spina bifida; cord reached to pelvis.
39	F.	Still-born.	Of an orange.	Posterior lobe of left hemisphere.	Effusion into ventricles.	Less than usual.	—
40	—	Still-born.	—	—	—	—	Spina bifida.
41	—	Still-born.	Large.	—	Effusion into ventricles.	—	—
42	F.	118 days.	Of a fœtal head.	—	—	—	Spina bifida.
43	M.	1 month.	Nearly that of the head.	Posterior lobes of cerebrum.	Sound.	—	—
44	—	51 days (operated on).	30 centimeters round.	Nearly the whole brain.	Softened and disorganised.	—	—
45	—	Fœtal.	—	—	—	—	—
46	—	Still-born.	—	—	—	—	Spina bifida.
47	M.	8 days.	43 centimeters long, 31 centimeters wide.	Posterior lobes of cerebrum.	—	—	—

State of Bones (at Hernia).	Other Malformations.	Reference to Author.
Large hole in occipital, continuous with foramen magnum.	Tumour and foetal membranes adherent together; body cleft in front from neck to navel; abdominal viscera and thoracic (except lungs), exposed; pericardium wanting; ventricles of heart communicating; ductus arteriosus absent; left supra-renal capsule wanting; right very small; cleft palate; club-feet; imperforate anus.	Otto, <i>Monstrorum sexcentorum Descriptio Anatomica</i> , 1841.
Occipital reduced to its foramen magnum.	Whole vertebral column greatly bent forwards; five right false ribs wanting; supra-renal capsules distended into two communicating cysts; fundus of uterus double; club-feet; cleft palate.	Ibid.
Hole above foramen magnum.	—	Ibid.
Hole above foramen magnum.	Atrophy of right supra-renal capsule.	Ibid.
Large hole in occipital, continuous with foramen magnum.	Fissure of soft palate; supra-renal capsules atrophied.	Ibid.
Hole in occipital, continuous with foramen magnum.	Supra-renal capsules atrophied; a cervical vertebra absent.	Ibid.
Hernia at the posterior fontanelle.	Umbilical hernia; velum palati cleft; fingers and toes grown together.	Ibid.
Hole in occipital at lambdoid angle.	Cervical vertebræ reduced to four; these ankylosed to one another.	Ibid.
Hole in right side of occipital.	Cleft palate.	Ibid.
Hole in occipital below lambdoid angle.	Eversion of the bladder.	D. J. Taylor, <i>London and Edinburgh Monthly Journal of Medical Science</i> , for 1842.
—	—	W. Lyon, <i>Med. Gazette</i> , for 1844.
Separation of the halves of the proral part of the occipital.	—	Forgemol, <i>Bull. de l'Acad. de Méd.</i> , 1844-5, p. 493.
—	Umbilical hernia of the liver, stomach, spleen, and intestines.	Vrölik, <i>Tabulæ ad illustrand. Embryogenesin Hominis et Mammalium</i> , 1844-9.
Hole in occipital, continuous with foramen magnum.	Neck apparently absent.	Ibid.
Hernia at the greatly enlarged posterior fontanelle.	Club-foot; suprarenal glands very large.	Ibid.; In Spring's paper in the <i>Mém. de l'Académie Royale de Médecine de Belgique</i> , 1854.

No.	Sex.	Age at Death.	Size.	Part of Brain protruded.	State of Cerebrum.	State of Cerebellum.	State of Spinal Cord.
48	F.	Still-born.	Size of head.	Posterior lobes of cerebrum and cerebellum.	Effusion into ventricles.	Atrophied.	Spina bifida.
49	M.	58 days (operated on).	Of a fowl's egg.	Left posterior lobe of cerebrum.	—	—	—
50	—	30 days.	9½ in. round.	The whole, except cerebellum.	—	—	—
51	F.	14 days.	2 in. across.	—	—	—	—
52	F.	2 days (operated on).	Enormous.	Left posterior lobe of cerebrum.	Effusion into ventricles	Atrophied.	—
53	M.	8 days.	—	Posterior lobes of cerebrum.	—	—	—

II. Of those occurring in

No.	Sex.	Age at Death.	Size.	Part of Brain protruded.	State of Cerebrum.	State of Cerebellum.	State of Spinal Cord.
54	—	4 days.	Very large.	Whole of cerebrum.	—	—	—
55	—	—	Two thirds that of head.	—	—	—	—
56	—	2 days.	Of a fowl's egg.	Great part of brain.	—	—	—
57	—	—	—	—	Right ventricle distended.	—	—
58	M.	14 days.	—	Anterior lobe of right hemisphere.	Atrophied; effusion into right ventricle.	Normal.	—
59	—	Some days.	Of the child's head.	Anterior lobes of cerebrum.	Effusion into ventricles.	—	—
60	—	11 weeks.	21 in. round.	—	—	—	—

State of Bones (at Hernia).	Other Malformations.	Reference to Author.
—	Cervical vertebræ reduced to two and split.	L. D. Le Roy, <i>Verhandelingen van het Genootschap ter bevordering van genees-en-heelkunde tot Antwerpen</i> ; anno viii, p. 253.
Hole above occipital protuberance.	—	A. Willems, <i>Ann. de la Société de Médecine de Gand</i> , for 1846.
Protrusion at the posterior fontanelle.	—	Ortwin Nægèlè, <i>Pr. Ver. Ztg.</i> , for 1849, p. 9.
—	—	Lussana, <i>Gaz. Med. Ital. fed. Lomb.</i> , 1851.
Hole above occipital protuberance.	—	De Lavacherie, <i>Bull. de l'Acad. de Brux.</i> , t. vi.
—	Suprarenal capsules very large.	Spring, <i>Journal de Méd. de Brux.</i> , 1853.

the Frontal Region.

State of Bones (at Hernia).	Other Malformations.	Reference to Author.
—	—	Oliv. Jacobæus, <i>Act. Med. et Philosophic., Hafniensia</i> , 1677.
Frontal and part of parietals absent.	Ureters and urethra absent; cleft palate; lips, chin, and external ear absent; arms and legs each only with one bone.	Gottl. Friderici, <i>Monstrum Humanum Rarissimum</i> , Lipsiæ, 1737.
Hole in frontal.	—	Saxtorph, <i>Collectanea Societ. Havniens.</i> , vol. ii, p. 280, 1775.
Opening between frontal and nasal bones. Separation of coronal portions of frontal.	—	Kelch, <i>Beiträge zur Patholog. Anatomie</i> , 1813.
Hole at glabella.	—	Osiander, <i>Goet. Gel. Anzeig.</i> , 1812, No. 139, p. 1377.
Separation of halves of frontal.	—	Bèclard, <i>Bull. de la Faculté de Méd.</i> , t. iii, p. 292, 1814.
—	—	Meiners und Benecke, <i>Langenbeck's Nosologie u. Therapie</i> , t. v, 3, p. 1395, 1822-25.

No.	Sex.	Age at Death.	Size.	Part of Brain protruded.	State of Cerebrum.	State of Cerebellum.	State of Spinal Cord.
61	—	Fortnight.	Of an apple.	Anterior lobes of cerebrum.	Softened.	—	—
62	—	Some days.	Of a nut.	Anterior lobes of cerebrum.	—	—	—
63	F.	Still-born.	8 in. round.	Anterior lobes of cerebrum.	Softened.	—	—
64	—	Still-born.	Of a chestnut.	—	—	—	—
65	F.	Still-born.	Of an orange.	Anterior cornu of left ventricle.	Effusion into ventricles.	—	—
66	M.	Fœtal.	Two, each the size of half a walnut, and one the size of a fist.	Anterior lobes of cerebrum.	Effusion into ventricles.	Small.	—
67	—	21 days.	Two, each the size of a pea.	Anterior lobes of cerebrum (?)	Meningitis.	—	—
68	F.	10 days.	Of a pigeon's egg.	—	—	—	—
69	M.	6 weeks.	—	Anterior lobes of cerebrum.	Meningitis and cerebritis of protruded part.	—	—
70	F.	6 months.	Of an olive, increased to that of a fowl's egg; before death.	—	Effusion into ventricles.	—	—

State of Bones (at Hernia).	Other Malformations.	Reference to Author.
Separation of coronal parts of frontal.	—	Breschet, Arch. Gén. de Médecine for 1831.
Hole between the right os unguis and frontal.	—	Ibid.
Separation of ethmoid and frontal. Frontal pushed upwards.	Uvula cleft.	Niemeyer, De Hernia Cerebri, Congenita, Halæ, 1833.
Separation of coronal halves of frontal.	—	Moreau, Dict. de Médecine. vol. xii, 1835.
Hole above nasal bones.	—	Otto; op. cit.
Large hole in frontal.	Left half of the body cleft from the axilla to the umbilicus; gall-bladder wanting; two supernumerary nostrils; cleft palate; a cutaneous appendix from the vertex of the head; left upper extremity atrophied, and four-fingered.	Ibid.
Deficiency of orbital plates of frontal and of roof of ethmoidal cells.	—	W. Lyon, Monthly Journal of Medical Science for 1842.
Crista galli absent. Ungual bones absent. Nasal and superior maxillary bones defective.	—	Schmitt, Med. Correspond. Bl. Bayerischer Aerzte, 1842.
—	—	Raynaud, Comptes Rendus de l'Acad. des Sciences, t. xxiii, p. 50, 1846.
Ethmoid shortened and depressed. Orbital parts of frontal depressed.	—	Dr. Clar, Zeitschr. der Gesell. der Wiener Aerzte, vol. vii, for 1851.

III. *Of those occurring in the*

No.	Sex.	Age at Death.	Size.	Part of Brain protruded.	State of Cerebrum.	State of Cerebellum.	State of Spinal Cord.
71	F.	30 days.	$\frac{1}{2}$ inch in diameter.	Left middle lobe of hemisphere.	Atrophy of left hemisphere; meningitis; cerebritis. Softened.	—	—
72	—	—	Voluminous.	—	—	—	—
73	F.	Still-born.	Large.	—	—	—	Left brachia swelling absent.
74	—	17 days.	—	—	—	—	Spina bifida.
75	F.	Still-born.	—	Nearly the whole cerebrum and cerebellum.	—	—	—

Parietal and Temporal Regions.

State of Bones (at Hernia).	Other Malformations.	Reference to Authors.
Squamous portion of temporal bone absent.	—	Billard, Tr. des Malad. des Enfants, 1828.
Separation of fronto-parietal suture.	Vast umbilical hernia, containing all the abdominal viscera and the heart; umbilical cord adherent to the head; club-foot.	Béclard, Bull. de la Faculté de Médecine, t. iii.
Bones of vault of cranium absent.	Placenta and the encephalocele connected by a ligament; right suprarenal capsule absent, left very small and cystic; left arm absent.	Otto; op. cit.
—	—	Dr. Camstock, Boston Journal, vol. xi, p. 241.
Left half of cranial vault absent; right half defective.	Adherence of brain to placenta and foetal membranes; thoracic and abdominal walls cleft, causing hernia of the heart, left lung, and all the abdominal viscera; left eye incomplete; hare-lip; cleft palate; left forearm and arm adherent; fingers of left hand reduced to one; club-foot.	Houel, Gazette des Hôpitaux, for 1850.

The preceding table contains 75 cases of this remarkable malformation, collated in every available instance with the works in which they were originally recorded.

The following is an analysis of some of the leading facts observed in these cases :

Congenital hernia of the brain occurs with pretty equal frequency in either sex. Of 39 cases 21 were males, 18 females.

The portion of brain protruded varies extremely in size, from that of a pea to dimensions surpassing those of the child's head. If the hernia occur at the occiput, the posterior lobes of the cerebrum and sometimes the cerebellum are protruded ; if at the frontal region, the anterior cerebral lobes. In some few cases (e. g., Cases 13 and 54) the entire brain had quitted its natural dwelling.

Meckel long since pointed out that the occiput was the chosen seat of encephalocele.¹ Of the 75 cases, 53 were in that position. The disease bears a marked analogy to spina bifida : indeed, in 16 of the 75 cases the two malformations were present together.

The cerebrum is rarely sound in these cases. Among 44 cases in which the state of this organ is specified, in only 5 cases was it healthy. In the remaining 39 cases it was either atrophied, softened, or, what is very commonly the case, effusion had taken place into its lateral ventricles.

In 19 cases the state of the cerebellum has been mentioned by observers, and it is not unworthy of remark that in 10 of these cases this organ was atrophied. It was so too in the case I have narrated.

As regards the duration of life, the cases admit of being divided into two categories, one in which the subjects of the malformation have lived for years, a second in which they have either been born dead or have survived but a brief

¹ 'Handbuch der Patholog. Anatomie,' Leipzig, 1812.

period after birth. Of the former class, there are six authentic instances on record.

(1) In 1774, M. Guyenot brought before the Academy of Surgery of Paris a man, 33 years old, who was born with a tumour on the left side of his forehead. This tumour measured $2\frac{1}{2}$ inches across, and pulsated. Around it could be felt a defect in the ossification of the frontal bone. The man's intellects were unimpaired; but there was a deficiency of power of the right arm.¹ (2) In 1813, M. Lallemand was about to operate on a girl, 23 years of age, for what appeared to be an ordinary wen on the back of her head. He had circumscribed the base of the tumour by a circular incision, when he detected the dura mater. The operation was at once left off; but the girl died within eight days from meningitis. The tumour proved to be an encephalocle. (3) Wedemeyer observed a case, in an idiot, 18 years old.² (4) Mr. Robert Adams relates an instance of hernia of the brain in a man, aged 20, in whom the tumour occupied "nearly the whole of the right half of his forehead."³ The same surgeon had (5) a little girl under his care, who was born with an encephalocle about the size of a hen's egg below the tuberosity of the occipital bone. The tumour was repeatedly punctured, and fluid drawn off from it. The child was seen well when six years old, without, however, the tumour having diminished in size.⁴ (6) Breschet met with a subject in the dissection room: an adult with a large tumour of the occiput containing the cerebellum.⁵

The following case has been often quoted as a case of

¹ 'Mémoires de l'Académie de Chirurgie,' t. v, in 4to, p. 863.

² 'Journal für Chirurgie und Angenheilkunde,' von C. F. v. Gräfe und Ph. v. Walther, vol. ix.

³ In the 'Dublin Journal for Medical and Chemical Science' for 1833, vol. ii.

⁴ Loc. cit.

⁵ In the 'Archives Générales de Médecine' for 1831.

congenital encephalocele; but appears to have been rather the consequence of the operation undertaken for its cure, than of congenital origin. J. N. Held had a young lady, aged 19, under his care for a congenital tumour of the forehead, the size of an apple. He opened it with a lancet. Some serum escaped and a pulsation was perceived. "Directly a small portion of brain rose up through the aperture in the os frontis and became prominent." The membranes of the brain were not injured in the operation, the aperture in the cranium closed, and the patient recovered in eleven weeks.¹ Equally equivocal is a case mentioned by Doepp, that, he states, lived several years, for the "out-growth" (Auswuchs) only sprang from the brain, to which it was but connected by the arachnoid, the tumours and brain being separable without tearing either.²

With the above isolated exceptions, all the cases of encephalocele belong to the second category, that is to say, are exceedingly short-lived. On this head the testimony of most observers agrees. M. Isidore Geoffroy Saint-Hilaire says "they are generally born alive, but die within a few days;"³ Dr. Montgomery that "most children so affected are either still born, or live but a very short time."⁴ Out of 68 cases which I have collected 32 were born dead. Mr. Taylor has recorded the case (No. 42 in the table) of a child who lived 118 days with a tumour the size of a foetal head. Dr. Clar one (No. 70) who lived upwards of six months. But in this latter instance the tumour was at first only the size of an olive, and never grew larger than a fowl's egg. The case I have narrated seems to stand alone in this, that while the tumour equaled in size the

¹ 'Dissertatio de Hernia Cerebri,' Giessæ 1777.

² 'Abhandlungen der Petersburger Aerzte' for 1842.

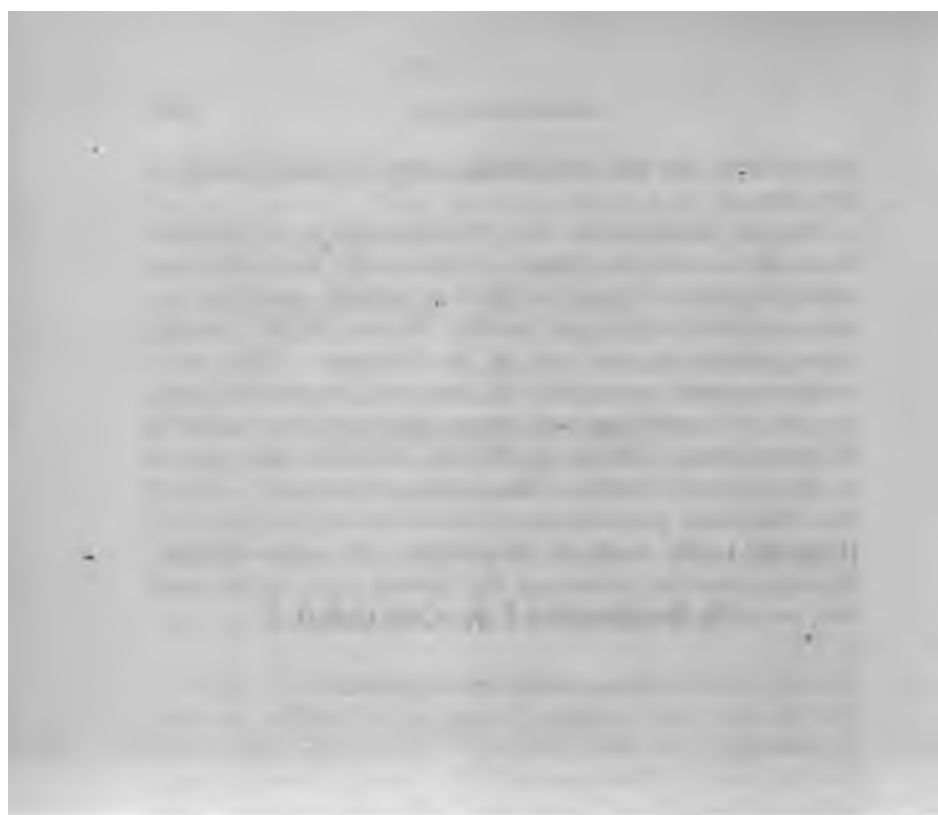
³ 'Traité de Tératologie,' par M. Isidore Geoffroy Saint-Hilaire, Paris, 1832.

⁴ 'The Cyclopædia of Anatomy and Physiology,' edited by Robert B. Todd. M.D. F.R.S., vol. ii, p. 320; article "Fœtus."

child's head, life was yet prolonged for the long period of 144 days.

For the alleviation of this disease surgery has hitherto been able to do but little. I have only met with two well-authenticated cases in which a surgical operation has been attended with a good result ; the one of Mr. Adams, above alluded to, and one of M. Richoux. The latter surgeon operated on a child ten days old, the tumour being the size of a fowl's egg and situate above the outer canthus of the right eye. He opened the sac, and sliced off a portion of the protruded brain. The patient recovered.¹ In all the other cases in which any operation has been undertaken, it has led to the death of the patient. It is probable that for any operation to succeed the tumour must be of small size, as in the two instances I have just quoted.

¹ 'Revue Médico-Chirurg,' p. 358.



AN ACCOUNT OF THE ARRANGEMENT
OF THE
MUSCULAR SUBSTANCE
IN THE
URINARY AND CERTAIN OF THE
GENERATIVE ORGANS
OF THE
HUMAN BODY.

BY
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SOME careful dissections of the muscular substance connected with the urinary and generative apparatus of the human body have made known to me many facts with which anatomists seem to be unacquainted; and the desire to add some of these to our common stock of knowledge, so that inaccuracy in our anatomical descriptions may be removed, has influenced me in making the present communication to the Royal Medical and Chirurgical Society. I do not purpose entering on the consideration of the nature of the muscular fibre, and the characters by which this may be

recognised where its existence is doubtful. The microscope will not, therefore, be needful to decide on the correctness of what I advance. With a scalpel and forceps, and a good eye, or, at most, with the aid of a watchmaker's eye-glass of an inch or an inch and a half focus, the following statements may be verified.

Of the Muscular Substance of the Urinary Bladder.

In the bladder there are three more or less perfect strata of involuntary muscular fibres; viz., an external or longitudinal, middle or circular, and internal longitudinal or submucous. These three are united by a mutual interchange of fibres, so that neither can be detached from the rest without cutting through these intercommunicating parts. In each layer, as in all muscular tissue,¹ the fibres are collected into bundles, and from these, offsets pass to adjacent bundles, producing thus a plexiform condition of the muscular web, or a network with meshes of varying size. (Fig. 1 and 2 a.) And as fleshy fasciculi connect the different strata, the fibres on the surface in the external layer become deep in the middle one, and the opposite; a like change in depth is observable between the submucous and the middle fibres. In consequence of this blending of the several layers, contraction of one will call into action the others, and thus the whole fleshy wall will take part in producing diminution of the viscus and expulsion of the urine.

The *outer layer* consists of fibres directed longitudinally from the apex to the base of the viscus, and is best marked below, in front of the neck. Above, the fibres end in tendons which are inserted, for the most part, into the fibrous peritoneal covering of the top of the bladder; but some are prolonged on the urachus, and end as the rest. Below they are differently arranged before and behind; in front some are attached to the back of the pubes by the

¹ The arrangement, structure, ending, &c., of the involuntary muscular fibre are elsewhere described by me.



anterior ligaments of the bladder, whilst others are continued over the sides and upper surface of the prostate as far as its apex, and end by tendon in its sheath; behind, the fibres terminate chiefly by blending with those of the deeper layer and the prostate, though in the female they reach also the fascia investing the lower part of the vagina.

The bundle of fibres connected with the back of the pubes is about half an inch wide, and has been described by other anatomists. Fleishy fibres radiate from it; some reaching the prostate and the neck of the bladder, where they cross those of the opposite side, and the remainder diverging to the base of that viscus. From the position of this fasciculus it is most advantageously placed for raising with its fellow the prostate and the neck of the bladder; and its action being, doubtless, to elevate those parts, the term *levator prostatae* might with propriety be applied to it.

The *middle layer* of circular fibres (fig. 1 and 2 *b*) is thickest towards the cervix vesicae, where it forms the ring called sphincter, and communicates largely with the submucous and external strata. Where the urethra begins it is continuous without any line of demarcation with the fleshy fibres of the prostate (fig. 1 *e*) in the male, and with a corresponding band of circular fibres in the female. The so-called sphincter is only part of the general muscular layer.

The *inner* or *submucous* stratum, alike in both sexes, is much thinner, and does not form so complete a layer as the others. In the lower third or half of the bladder, the fibres are longitudinal in direction, and of tolerably uniform thickness (fig. 1 and 2 *c*); but higher up they are thin, and become oblique and scattered on the mucous membrane. Behind, below the openings of the ureters, this stratum receives an accession of fleshy fibres from the muscular coat of those tubes (fig. 2 *r*). At the neck of the bladder this stratum is continued into the submucous coating around the urethra; and as the fibres pass from one part to another they form the projection called "uvula vesicae." The existence of this layer may be shown by dissecting it

from below upwards, after the manner indicated in fig. 1 and 2; the bladder having been previously hardened in a strong solution of salt, and then distended with cotton wool or tow, introduced through a small opening in the top.

The ending of the ureters may be traced by taking away the two outer vesical strata and part of the prostate, as in fig. 2. These canals pierce the outer and middle strata of the fleshy wall of the bladder, and the fibres of their muscular coat are disposed in both sexes as in fig. 2; viz., the internal, the most numerous, are directed transversely, and unite with the corresponding fibres of the opposite ureter; whilst the remainder join the submucous muscular layer of the bladder, and are directed obliquely downwards over the "triangular space" to the submucous stratum of the urethra.¹

Muscular Substance of the Prostate.

The prostate is essentially a muscular body, consisting of circular or orbicular involuntary fibres, with one large central hole for the passage of the urethra; and another smaller, oblique opening, directed upwards below the former, for the transmission of the common ejaculatory seminal ducts to the central urinary canal. The few longitudinal fibres on the upper surface of the prostate, which are derived from the external layer of the bladder, can scarcely be said to form part of that body.

Its circular fibres² are directly continuous behind, without any separation, with the circular fibres of the bladder; and in front a thin stratum, about one thirtieth of an inch

¹ The ending of the ureters has been differently described by the late Sir Charles Bell, in vol. iii of the Transactions of this Society. A fleshy bundle, which he called muscle of the ureter, is described as descending from the uretral opening towards the neck of the bladder, where it joins the like part of the other side; and the two conjoined are said to be inserted by a tendon into the middle lobe of the prostate.

² The thickness, dimensions, and topographical relations of the prostate will be found described in the common text-books.

thick, is prolonged forwards from it around the membranous part of the urethra¹ (fig. 1 e), so as to separate this tube from the surrounding voluntary constrictor muscle. These facts seem to show the inappropriateness of the older view respecting the distinctness and glandular nature of the prostate, and indicate its being a portion of a muscular layer, which surrounds the intra-pelvic part of the urethra, and is continuous without interruption with the circular fibres of the bladder. Within, and quite distinct from the circular fibres, lies the tube of the urethra, incased by its submucous layer of longitudinal fibres (fig. 1 d). Towards the lower and outer aspects, the fibres are less firmly applied together, especially where the vessels enter; and they appear to be superadded to those which join the coat of the bladder.

As only so small a portion of the prostate is glandular, the propriety of calling that body a gland is rendered doubtful; for the small secreting glands contained in it are but appendages of the mucous membrane, which project amongst the muscular fibres in the same way as the other glands of the urethra extend into the surrounding submucous tissues. The glands are situate towards the base of the prostate, and more are below than above the urethral tube, especially in the part called middle lobe; their largest ducts enter the urethra opposite the opening in the circular fibres for the common ejaculatory seminal ducts.

In the female the urethra corresponds with the intra-pelvic part (prostatic and membranous) of that of the male, and is surrounded in all its length by circular involuntary muscular fibres, which are external to the submucous, and are continued, as in the male, into the middle stratum of the bladder. Thus, the posterior third of the urethra of the

¹ Professor Müller has described a layer of voluntary circular fibres around the membranous part of the urethra; but he considers it to form part of the constrictor urethræ muscle outside, and he does not show its connection with the prostate and bladder. See the treatise of J. Müller, 'Ueber die Organischen Nerven der erectilen Männlichen Geschlechts Organe des Menschen,' &c., Berlin, 1836.

male differs chiefly from the tube of the female in having the surrounding involuntary circular muscular fibre extremely developed at one spot in connection with special functions, and in being provided at the same spot with large secreting glands, for the purpose of increasing the quantity of the seminal fluid.

From the above-given anatomical facts, we may conclude that the prostate is less of a glandular than a muscular body, and is only a largely developed portion of the circular muscular layer that invests all the urethra behind the bulb or the spongy portion. The existence, too, in the female of a thin muscular stratum in the corresponding position gives support to the view of its muscular office. As the prostatic enlargement includes only part of the muscular stratum on the urethra, I would propose the name *orbicularis vel sphincter urethræ*¹ for both the prostate and the prolongation around the membranous portion of the urethra; whilst I would confine the old term prostate (without the word gland) to the thickened and more powerful part near the neck of the bladder. This orbicularis may be considered as only an advanced portion of the circular layer of the bladder, though it must have the power of acting independently of the vesical fibres, as, for instance, in the propulsion of the seminal fluid. Its chief office will probably be, to hurry on the semen, and deliver this into the grasp of the voluntary muscular fibres of the constrictor urethræ, which are external to it along the membranous part of the urethral tube.

Submucous Fibres of the Urethra.

A submucous stratum of longitudinal muscular fibres surrounds the urethra throughout its whole length, and is continued behind into the submucous layer of the bladder. It is strongest around the first third of the urethra (that

¹ Professor Köl liker applies the term *sphincter prostate* to some of the more internal circular fibres. He says the other fibres radiate from the centre to the circumference. ('Mikroskopische Anatomie.')

next the bladder), especially so in the prostate (fig. 1 and 2), and becomes gradually thinner as it proceeds towards the end of the penis; much fibrous is intermingled with the muscular tissue. At the fore part of the urethra its fibres end in tendons in the usual way, many of these blending with the submucous fibrous tissue. At the hinder third, that part embraced by the circular fibres of the orbicularis urethræ, many longitudinal fibres become oblique in direction, and are applied to those of the orbicularis. And at the neck of the bladder, other fibres join the circular stratum of that viscus, as is represented in fig. 1 and 2. In the prostatic part of the urethra, the central median crest or ridge (*crista seu veru montanum*) is formed by a bundle of this layer, whose longitudinal fibres separate to enclose the opening of the vesicula prostatica (*sinus pocularis*); and in the same spot the submucous stratum is joined by muscular fibres that accompany the ejaculatory ducts. No circular fibres have been recognised by me in the submucous layer of the urethra; where such external fibres appear after the removal of the longitudinal, they belong to other structures, viz., to the orbicularis muscle in the posterior third, and to the septum corporis spongiosi (to be afterwards referred to) in the anterior two thirds of the urethra.¹

In the urethra of the female, the submucous fibres are like those in the male, and have a similar position and arrangement, blending behind with the circular fibres of the urethra and bladder; but as there are not any seminal ducts in this sex, the accessory bundle in the male is wanting. About a quarter of an inch from the anterior opening of the tube, the longitudinal fibres are collected into bundles, between which are openings of the submucous glands arranged in lines.

¹ I have not met with the circular fibres in this layer, which Professor Kölliker describes (*Mikroskopische Anatomie*). Still less could I verify the statements of Mr. Hancock respecting it. (*Lettsomian Lectures*, 'Lancet,' 1852.) Those who are acquainted with the descriptions of the writers referred to, will perceive that I have not borrowed my account from them.

Muscular Covering of the Vesiculæ and Vasa Deferentia.

A muscular layer covers and partly surrounds the vesiculæ seminales and the ending of the vasa deferentia. It will be brought into view, when the bladder is placed upside down, by removing carefully from the seminal vesicles their sheath of the recto-vesical fascia. It consists of one layer of longitudinal and another of transverse fibres; and the whole might be named, from its office, *compressor vesiculæ et ductûs seminis*.

The transverse fibres are the more superficial in the inverted position of the part; some stretch over the vesiculæ, and are inserted at each side into the investing fascia; but others, and these are the most numerous, reach only as far as the outer border of the vasa deferentia: by this arrangement both the parts of the seminal apparatus may be compressed. This stratum is thickest near the prostate, and joins in front the circular fibres of that body.

The longitudinal fibres are beneath the others, and form a less extensive plane. For the distance of half an inch behind the prostate, they give rise to a continuous fleshy layer over the seminal apparatus; and from this, fibres are continued forwards and backwards. The anterior offset surrounds the common ejaculatory ducts, and joins the sub-mucous layer of the urethra in the prostate; and the posterior is continued along the vasa deferentia for a short distance, as well as along each vesicula, where it is strongest at the outer and inner margins.¹

This muscular layer will compress and shorten the vesiculæ seminales, and the lower dilated ends of the vasa

¹ Professor Kölliker gives the following description of this structure: "Externally the vesiculæ are surrounded in part only by a membranous, and, in part, by an evidently muscular sheath, as at the hinder surface. This enters between the different windings of the canal uniting them together, and at the lower end passes as a broad muscular band from the one vesicula to the other." *Mikroskopische Anatomie: zweiter band*, p. 405.

deferentia. If the bladder is distended during the contraction, giving, in this condition, support to the seminal organs, the muscle will be enabled to act much more efficiently in the expulsion of the semen.

Sheaths around the Spongy Structure of the Penis.

The sheath investing the spongy material of the corpora cavernosa is commonly described as being composed of fibrous tissues, like those in tendons, without any special arrangement, except that they are said to be mostly longitudinal. My dissections demonstrate two layers of fibres in it, with a constant arrangement, one being superficial and having longitudinal fibres, the other deep, with transverse fibres, as in the wall of the alimentary tube. Moreover, the fibres have the same arrangement as in involuntary muscle, that is to say, they have a net-like disposition with small meshes.

In the outer stratum, where the fibres are directed longitudinally in bundles $\frac{1}{30}$ th to $\frac{1}{30}$ th of an inch wide, meshes about $\frac{1}{4}$ th of an inch in length are left between the offsets that pass from bundle to bundle. (See fig. 3.) Towards the front of the penis the bundles and the meshes are smaller; and in the crus the bundles are whiter, apparently from a greater mixture of fibrous tissue.

In the inner stratum, which is rather thinner than the other, the fibres are disposed circularly or transversely, though with the plexiform arrangement before described; and they will be best seen by opening the sheath and removing the spongy substance, as in fig. 4. Passing circularly around the cavity, the fibres blend in the middle line with those of the opposite corpus cavernosum, and construct in this way the imperfect septum (sept. pectiniforme) along the centre of the penis. (fig. 4 m.)

Only circular fibres enter into the composition of the septum between the cavernous bodies, as before described; and the fissures in it, corresponding with meshes in the other

parts, are here greatly enlarged to permit the free communication of the vascular or erectile tissue at opposite sides.

Where the cavernous bodies terminate in points anteriorly, small bands are continued into the glans penis.

The sheath of the corpus spongiosum urethræ resembles that of the cavernous bodies in incasing the vascular substance, and in possessing like them a central partition; but it has only one layer of fibres. It is formed of thin circular or transverse fibres, as is the deeper layer of the corpora cavernosa, but near the bulb some of them are oblique. Towards the front of the penis the place of longitudinal fibres is supplied by the "fascia penis."

The septum is connected with the surrounding sheath, and reaches vertically, as in the cavernous bodies, from the upper to the under aspect of the corpus spongiosum. As it crosses the included space it incases the urethral tube with the submucous layer, a piece being continued on each side. Its length below the tube of the urethra, and the way in which it surrounds this canal, are seen in fig. 1 *f, g*. This partition exists throughout the whole extent of the corpus spongiosum, though towards the front it is very imperfect, resembling in this respect the representative part in the corpora cavernosa. In a favorable body, I have traced it from the bulb to the glans, but its connection with the outer sheath is very slight in front; commonly it will not be traceable as a distinct partition much more than two inches from the bulb. It is composed of vertical fibres with minute clefts, as is the septum of the corpora cavernosa. All that part of the glans penis which is reflected back on the cavernous bodies above the tube of the urethra, wants the median partition.¹

The sheaths surrounding the vascular or erectile structures of the penis are, therefore, alike in their texture; but one is composed of two strata with fibres taking different directions,

¹ Mr. H. Thompson, in a 'Treatise on Stricture of the Urethra,' calls in question the fact of the septal process being continuous with the sheath; but I think he will allow the existence of this junction if he seeks it in the way represented in the drawing.

whilst the other has only a circular layer. Across the space included by the sheath in both the cavernous and spongy bodies is placed an imperfect partition, which is formed of circular fibres.

EXPLANATION OF THE FIGURES.

All the figures have been taken by the skilful hand of Mr. Ford from my own dissections.

FIG. 1.—For this figure the outer and middle strata of the muscular coat of the bladder, and the muscular layer around the prostatic and membranous parts of the urethra, were cut through in the preparation, so as to show the submucous stratum of the bladder and urethra. The corpus spongiosum was cut into, and the contained spongy material removed for a short distance to lay bare the septum. The same letters are used to indicate like parts in fig. 1 and 2.

a. External or longitudinal muscular layer of the bladder.

b. Middle or circular.

c. Internal or submucous. Many of its fibres are shown entering the circular fibres of the middle stratum.

d. Longitudinal submucous fibres of the urethra as they pass through the prostatic and membranous parts of that tube, internal to and separate from the circular layer *e*. At the neck of the bladder some are represented cut, as in the dissection, and others blend with the circular fibres.

e points to the circular fibres around the hinder third of the urethra, which I have named *orbicularis urethræ*: their continuity, behind, with the circular fibres of the bladder appears in the drawing.

f. The septum of the corpus spongiosum: the way in which this encloses, and shuts out the urethra with its longitudinal submucous layer from the spongy tissue, is referred to by *f*. Some apertures have been made in the septum to show the space on the other side: the piece of whalebone, introduced into the left half of the corpus spongiosum, appears through the apertures.

h. Corpus spongiosum urethræ.

k. Corpus cavernosum penis.

l. Ureter of the right side.

FIG. 2.—In the dissection here represented, after the separation of the vesiculæ seminales and vasa deferentia, which have been drawn forwards, the two outer strata of the muscular coat were taken away over the triangular space at the base of the bladder, in order that the expansion derived from the ends of the ureters might be seen joining the submucous layer. The lower half of the prostate was cut away in greater part, and the submucous layer of the urethra laid bare.

a, b, c. The different muscular strata of the bladder as in fig. 1.

d. The layer around the urethra in the prostate: the central ridge corresponds with the prominence in the floor of the urethra. Some of the fibres join the circular of the bladder, while others enter the deepest or the submucous layer of that viscus.

n. Vesicula seminalis and the vas deferens joining to form the common ejaculatory duct.

p. Membranous part of the urethra.

l. End of the ureter: some fibres from it, *r*, are seen joining those around the urethra, while others enter the submucous muscular layer of the bladder.

FIG. 3.—The longitudinal fibres and the meshes of the outer stratum of the corpus cavernosum are here indicated. The part selected for the drawing was not far from the crus penis.

FIG. 4.—The piece for the drawing fig. 3 was cut open, and the spongy tissue removed to make the preparation for fig. 4. It illustrates the circular arrangement of the inner layer of fibres of the corpus cavernosum; *m* points to the formation of the septum pectiniforme by those fibres.

ON
MERCURIAL FUMIGATION
IN THE
TREATMENT OF SYPHILIS.

BY
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Soon after the recognition of the venereal disease in Europe at the end of the fifteenth century, fumigations of various sorts were employed in its treatment. Among these mercurial fumigations soon held the first rank. But the want of precise knowledge of the nature of the substances used, rendered this mode of treatment liable to serious inconveniences; and the cumbrous nature of the apparatus employed, tended still more to bring it into disuse. Accordingly, the employment of fumigations in venereal diseases was discontinued, so soon as other modes of treatment more certain in their results, and of more easy application, were discovered. During a period of three centuries following, the attempts made to introduce mercurial fumigations were not more successful, owing to the want of care in their employment, and the defective nature of the apparatus used.

In 1776, the Chevalier Lalouette published what he

called a new method of treating the venereal disease by fumigations. His method consisted in placing the patient in a closed box, from which the patient's head projected, so as to allow the mercurial vapour to act upon the whole surface of the body, while the respiration was not interfered with. The mercurial preparation used was a kind of calomel, which, by repeated sublimation with iron filings, was so far deprived of its muriatic acid as to be in part reduced into running quicksilver.¹ This preparation was placed on a heated metallic plate at the bottom of the box, and was immediately converted into smoke, which surrounded the patient's body.

The mode of preparing this powder was troublesome and expensive. Abernethy made it in a much more simple way by adding two drachms of liquor ammoniæ to six ounces of distilled water. Four ounces of calomel were then thrown into this mixture, and the whole shaken up together. The powder was afterwards separated by a filter and dried. It was then of a gray colour, and contained a good deal of quicksilver in its metallic state.

Mr. Pearson tried Lalonde's apparatus. He found that the patient's gums became turgid and tender very quickly, and that the local appearances of disease were sooner removed than by the other modes of introducing mercury into the system. The quantity of mercury used, rather than its effect on the patient's system, was considered, in Mr. Pearson's day, as the essential circumstance to be regarded in the treatment of syphilis; and accordingly he remarks:—"it is extremely difficult to introduce a sufficient quantity of mercury into the animal frame (by fumigation) so as to secure the patient against the hazard of a relapse."²

In attempting to introduce the quantity imagined to be requisite, Mr. Pearson found "that it induced a considerable debility in a short space of time, that a ptyalism was often excited rapidly;" and that, consequently, the remedy which

¹ Abernethy's 'Surgical and Physiological Essays.'

² Pearson's 'Observations on the *Lues Venerea*,' p. 123.

acted so powerfully on the animal frame had often to be discontinued.

The fumigating machine used by Lalouette, Mr. Pearson regarded as a very convenient one, but as no novel invention, as it differed in no material circumstance from that described by Nicholas de Blegny, in the year 1683.

In 1824, M. Rapou published two volumes on fumigations as employed in various diseases. The mercurial preparations which he recommends are—1st, cinnabar; 2d, Lalouette's mercurial powder, which consists of a mixture of mercury and clay; 3d, calomel; and, 4th, corrosive sublimate.

The quantity used of the three first he recommends to be from "a quarter to half an ounce each time." That of the last, he says, should not exceed five or six grains, and may be volatilized with the vapour of water by means of a modification in the fumigating apparatus. M. Rapou had only witnessed salivation produced in three instances, and these were of a very short continuance, and yielded to the use of ordinary baths. He recommends, in certain cases, the mercurial fumigations to be used with steam which, as he says, calms the system, softens the skin, and does not prevent the absorption of the mercury (vol. ii, p. 395).

All the methods of applying the mercurial vapour above noticed required the use of an apparatus, and were, consequently, attended by expense and loss of time. For these and other reasons before mentioned, the mode of treatment never became general. It has, however, again been revived within the last few years in a way in which its effects are easily regulated, and which requires no apparatus that every one cannot generally command. Mr. Langston Parker recommends that the patient should be placed on a chair, and covered with an oil-cloth, lined with flannel, and supported by a proper framework. Under the chair are placed a copper bath, containing water, and a metal plate on which is put from one to three drachms of the bisulphuret of mercury, or the same quantity of the gray oxide or the

binoxide. Under each of these a spirit-lamp is placed. The patient is thus exposed to the influence of three agents;—heated air, common steam, and the vapour of mercury, which is thus applied to the whole surface of the body in a moist state.

Practically the inconvenience of this mode of treatment arises from the gray oxide being of very uncertain composition, as procured at the shops; and from the bisulphuret being often adulterated. Moreover, both these preparations require more heat than is furnished by a common spirit-lamp for their conversion into vapour.

The preparation usually kept by chymists as the gray oxide, is the protoxide, and is of a greenish or blue colour. When heat is applied to this, it becomes converted into the red deutoxide; and if the heat be further increased, the oxygen is driven off, and the metallic mercury either left, or raised in vapour. The action of this substance is uncertain and unsatisfactory. A preparation under the same name, of a lighter or gray colour, may be obtained from some chymists, which volatilizes well at the ordinary temperature of a common spirit-lamp, and produces its effect with much certainty upon the patient's constitution. The gray colour of this powder depends upon the admixture of a certain proportion of calomel with the protoxide; and the comparative low temperature at which it volatilizes depends upon the same cause. If this last preparation could always be procured, containing the same amount of calomel, it would answer very well for the purpose of mercurial fumigation; but obtained at different shops, and even at different times at the same shop, it is found to vary materially in its composition.

Finding from experience that it was the light coloured oxide alone which volatilized, and produced its effects upon the patient's constitution, and having reason to believe that the light colour depended upon the presence of calomel, I performed a series of experiments with calomel alone, or mixed in a certain proportion with the gray oxide. The



general result of these experiments has been, to satisfy me that, for the purposes of mercurial fumigation, five or ten grains of calomel alone is, in ordinary cases, quite sufficient ; and that when the gray oxide is used, the admixture of a few grains of calomel will facilitate its sublimation, and insure its medicinal action.

In Lalouette's apparatus, according to M. Rapou (vol. i, p. 40), the mercury was raised in a metallic state (the gray oxide being first converted into a deutoxide, and the oxygen then being in great part or altogether driven off). The preparation used was therefore of uncertain composition, and its effects must necessarily have varied also. Now calomel is not acted upon either by heat or the vapour of water, and, consequently, can be used with comparative certainty both as regards its composition when volatilized, and its physiological effects.

Upon making comparative trials with the calomel alone and combined with steam, it was found to act more certainly and with greater regularity in the latter case.

The plan which I have adopted is very simple. Two small lamps are procured in which the methylated spirit (much cheaper than spirits of wine) is used ; over the first lamp is a thin metallic plate, upon which the ten grains of calomel are placed ; over the second lamp is a small cup of hot water. A small cane bottomed chair is placed over the lamps, and the patient sits upon it. He is then enveloped, chair and all, in a blanket ; at the expiration of a quarter of an hour or twenty minutes he rolls himself up in the blanket and goes to bed.

For patients to whom it may not be convenient to procure the spirit-lamps, the mode of proceeding may be varied as follows : The patient is directed to heat a thick tile in the fire ; this is then put into a night-stool, and a gallipot full of warm water placed upon one corner of it. The calomel powder is then sprinkled over the rest of the tile, and the patient sits over it, being enveloped, as before, in a blanket. This mode of applying the vapour is very con-

venient in cases of affections, either primary or secondary, of the generative organs. It is not necessary in either case that the patient should breathe the mercurial fumes. It is remarkable how soon the patients' systems are brought under the influence of the mercury by this simple means; and, according to my experience, how effectually it acts in cases both of primary and secondary syphilis. Its great advantage, however, consists in the very little constitutional disturbance produced, and in the avoidance of those symptoms of irritation and debility, both mental and physical, which the prolonged internal use of mercury is so apt to occasion. The mercurial action, when the medicine is introduced through the skin, may be continued for nearly any length of time that may be necessary; and may be repeated as often as may be convenient, without injuring the patient's constitution.

The small quantity of calomel which it is requisite to use at each fumigation, is probably one reason why mercury in this form may be used with such comparative impunity.

The following table presents a view of the first cases in which the gray oxide alone, and in combination with calomel, and calomel alone, were made the subject of comparative experiment.

TABLE of Cases of Syphilis treated by Mercurial Fumigation in the Lock Hospital.

No.	Male or Female.	Age.	Disease.	Preparation and Quantity of Mercury Used.	Time at which Mouth became affected by the Fumigation.
1	M.	30	Secondary syphilitic disease.	Gray oxide, probably two drachms.	Mouth very sore with three fumigations.
2	M.	29	Secondary syphilitic disease.	Gray oxide.	5 days.
3	M.	34	Tertiary syphilis.	Gray oxide.	4 days.
4	M.	23	Secondary syphilis.	Gray oxide, two drachms every night.	4 days.
5	M.	21	Secondary syphilis.	Gray oxide.	4 days.
6	M.	26	Syphilitic lepra.	Gray oxide.	4 days.
7	M.	22	Syphilitic tubercles and blotches.	Gray oxide.	8 days.
8	M.	28	Tertiary syphilis.	Calomel, 15 grains, every night.	7 days.
9	M.	46	Secondary eruption; tubercle.	Calomel, 15 grains, every night.	6 days.
10	M.	20	Indurated primary sores.	Calomel, 15 grains, every night.	2 days.
11	M.	25	Indurated primary sore. Secondary eruption.	Calomel, 10 grains, every night.	10 days.
12	M.	28	Tertiary syphilis.	Calomel, 5 grains, every night.	8 days.
13	M.	31	Affection of lungs succeeding secondary syphilis.	Calomel, 5 grains, every night,	4 days.
14	M.	29	Scaly secondary eruption.	Calomel, 5 grains ; and gray oxide, 10 grains, every night.	2 days.
15	M.	22	Condylomata on scrotum and thighs.	Calomel, 10 grains, every night.	6 days.
16	M.	22	Indurated primary sore. Syphilitic psoriasis.	Calomel, 10 grains, every night.	6 days.
17	F.	20	Primary syphilis.	Calomel, 15 grains, every night.	5 days.
18	F.	19	Primary syphilis ; condylomata.	Calomel, 10 grains, every night.	4 days.
19	F.	19	Indurated primary sore.	Calomel, 5 grains ; and gray oxide, 10 grains, every night.	4 days.
20	F.	16	Indurated sore ; condylomata.	Calomel, 10 grains, every night.	8 days.

To prolong this table would be only to repeat the evidence upon the same point which subsequent experience in a large number of cases has fully established; namely, that a patient's system can be as readily influenced by a small quantity of calomel in vapour as by a large quantity of the gray oxide, or of the bisulphuret of mercury.

In consequence of the calomel not being liable to be decomposed either by heat, or by the vapour of water, its action is comparatively constant, and its influence upon a patient's system may be regulated with great nicety. From the small quantity used, there is little fear of producing salivation, while its effects upon syphilitic disease, in its various forms, are all that can be expected from any mode of treatment by mercury. The essential difference in the mode of using the vapour of calomel now recommended, and any former trials, that have been made, with this preparation, consists in the temperature used for the purpose of volatilizing it. If the temperature be high, as is necessary in other forms of mercurial fumigation, the whole of the calomel is at once converted into vapour and dissipated in a few moments. At a lower temperature, which can be much more easily obtained in private houses, the operation is prolonged, and time afforded for the vapour to produce its effect upon the skin.

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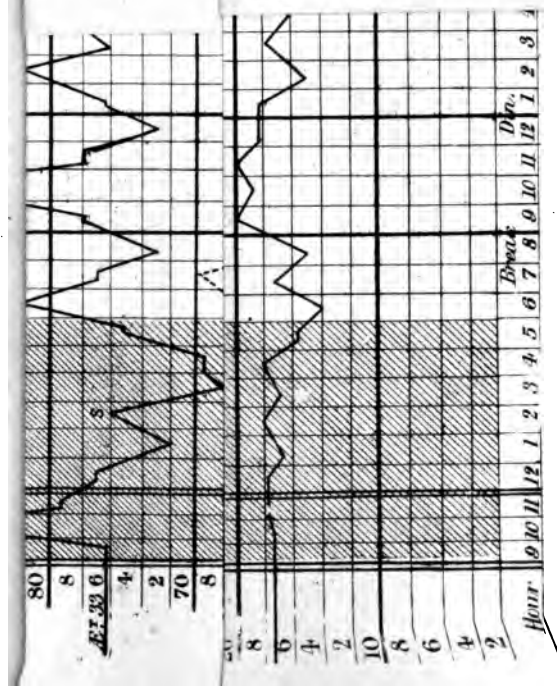
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Wound of abdominal parietes	23





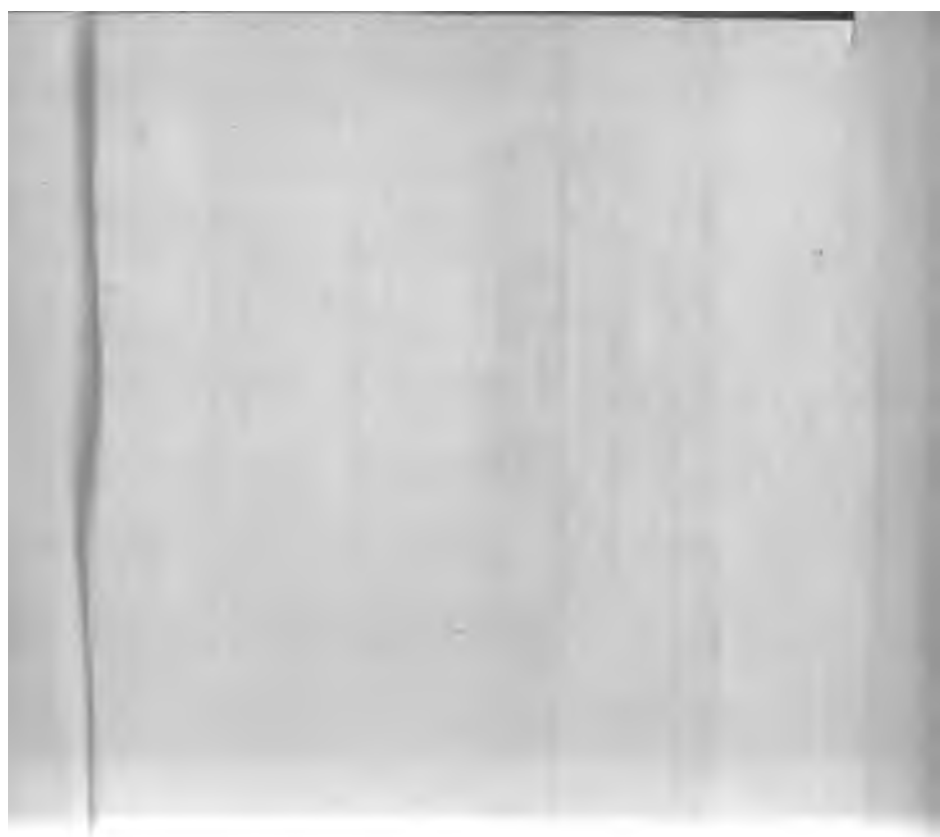
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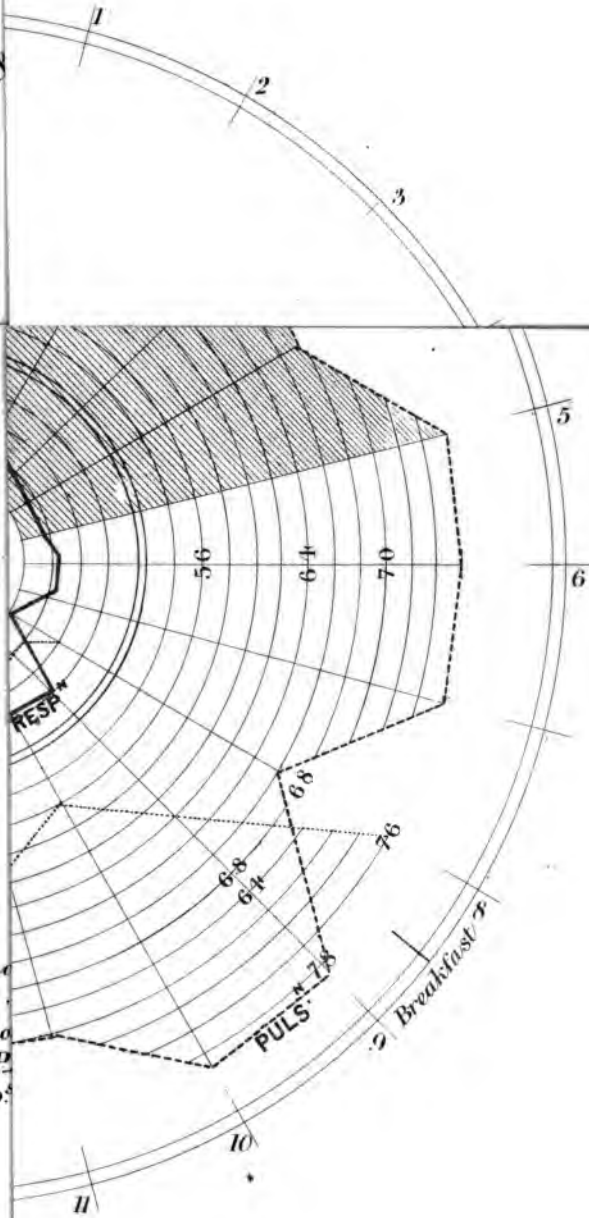
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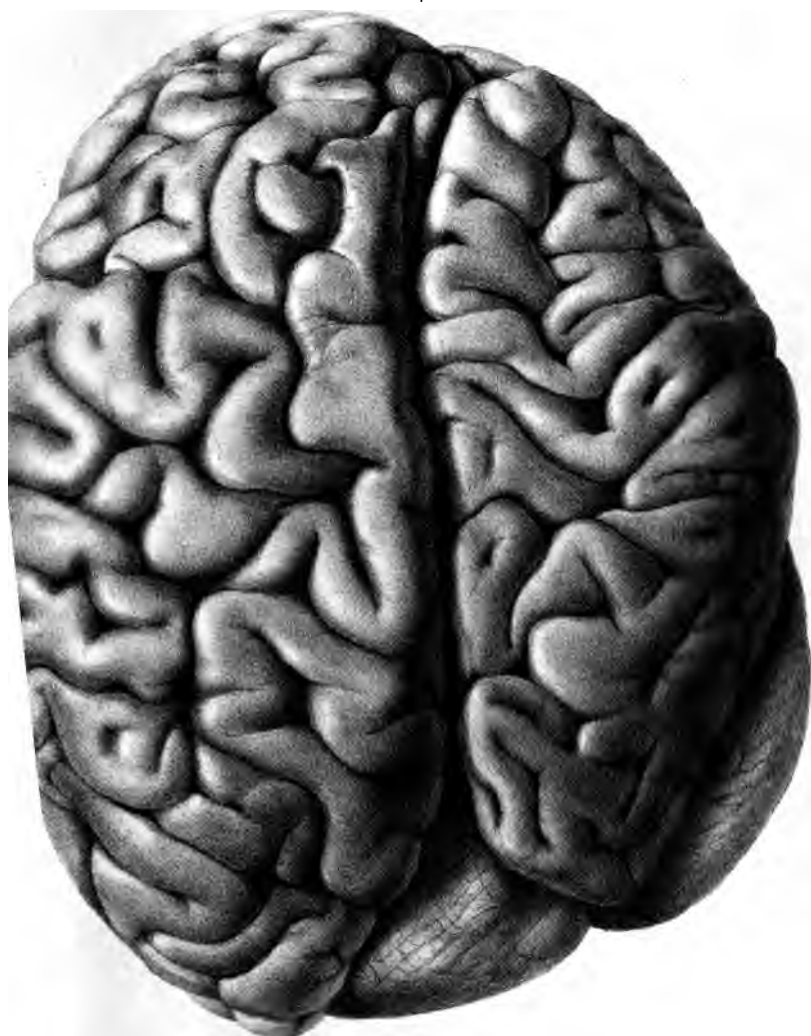


Early Pulsation 8
in
Health

Dr Edward Smith
Nov^r 1855.

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Scale of Inches for the above subject



Pl 1.

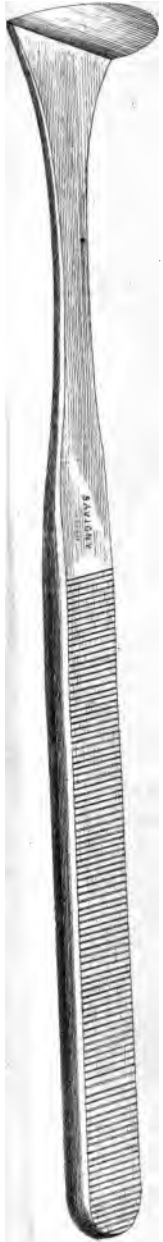


Fig 5.

CH Ford del

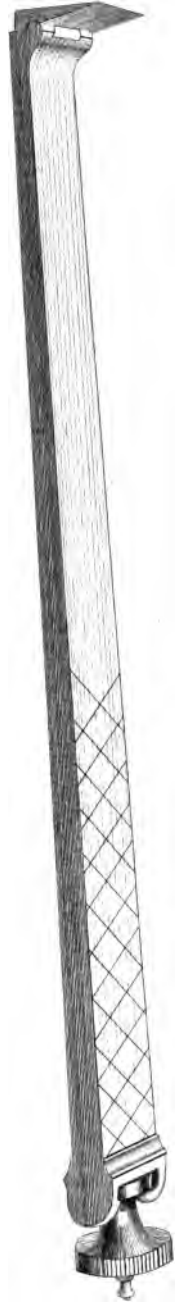
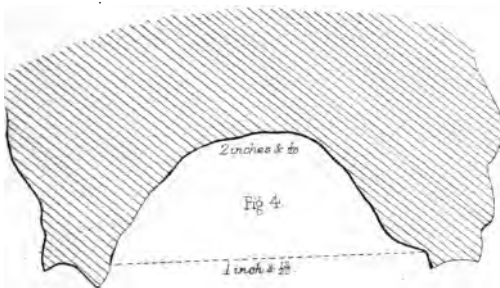
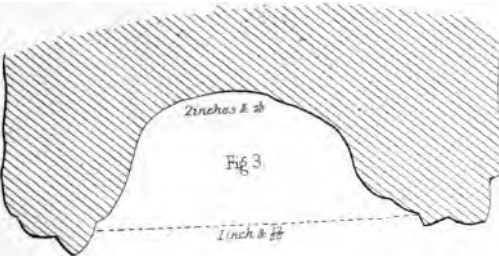
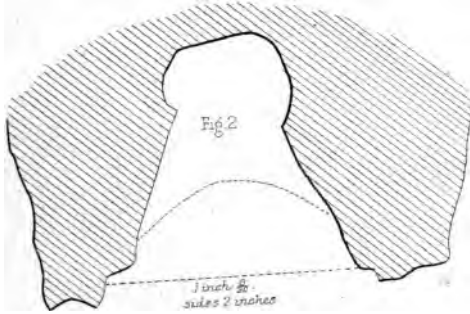
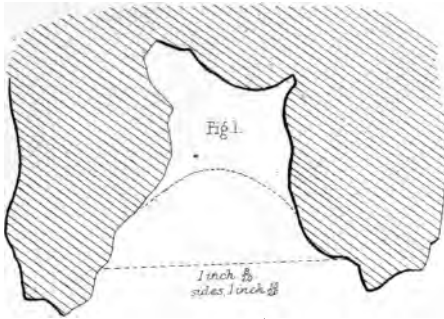
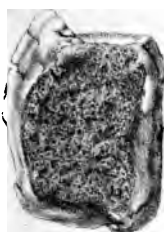


Fig 6.

H Wilson del

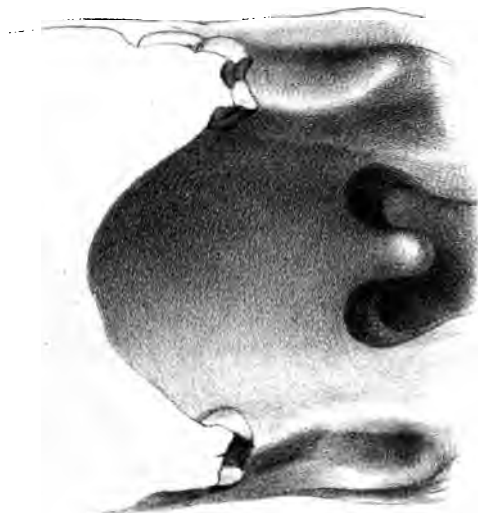




Under surface of inner condyle of femur,
the detached portion of articular cartilage, seen on both surfaces



Fig. 1.



G. H. Ford, Del.
W. West, Sculp.

Section to show the arch in a perfectly developed mouth.

Fig. 2.



Section to show that the arch of the palate is almost natural. The fissure is confined to the palate bones & soft palate.

W. West, Sculp.

1

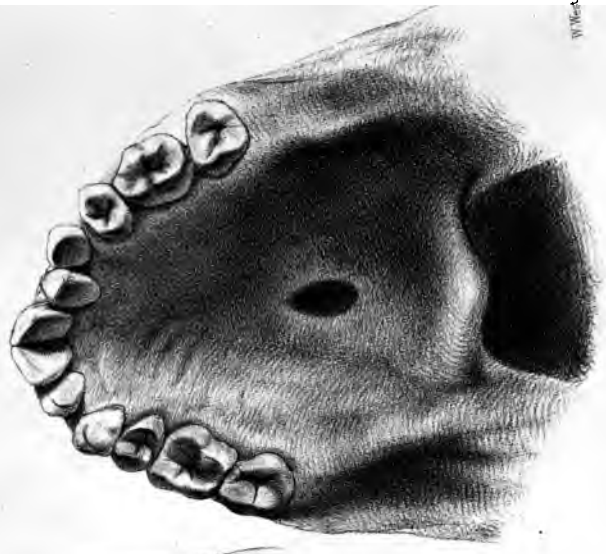
Fig. 1



The same case, as represented by
Fig. 1, after the first operation

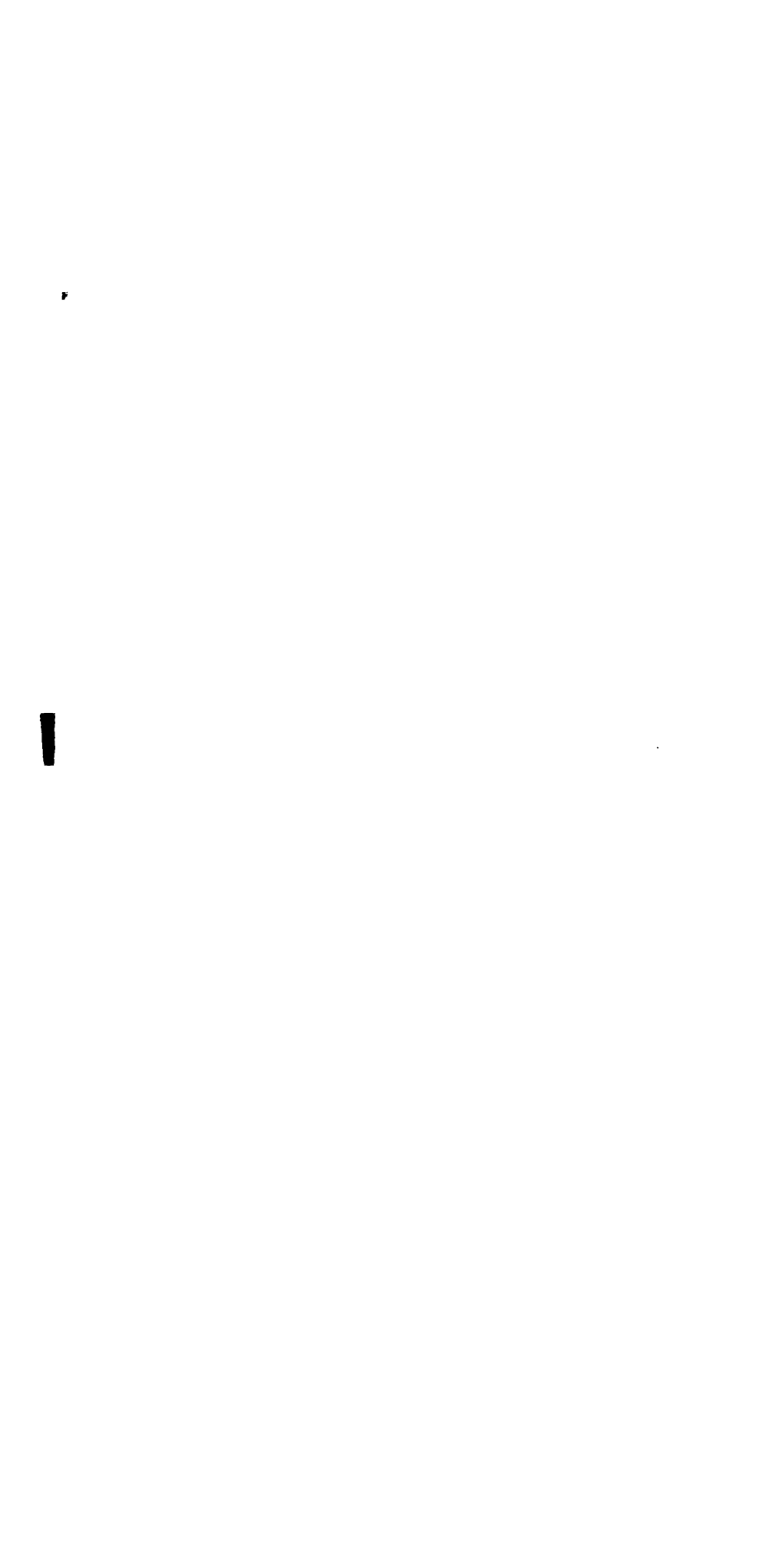
H. Wilson del.

Fig. 3



The same, after a second operation

W. West s.



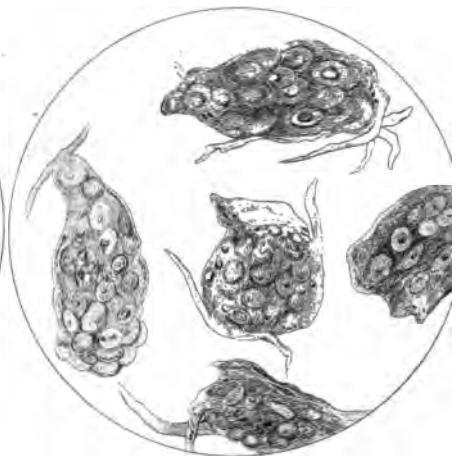


Appearance of the mouth with false lip adjusted.

1

Fig 1

Fig 2



2 Myeloid cells, some of which are furnished with caudate prolongations
400 diam.

Myeloid Tumour of the Tibia

Fig 3

Fig 4

Fig 5



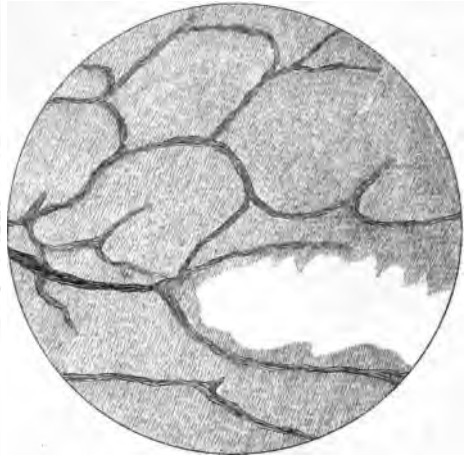
400 diam.

1 Fibrine with included Lymph Corpuscles from the interior of one of the cysts. 2 Nucleated spindle shaped fibres, from the solid matter of the Tumour.

3 Myeloid cells from the solid matter of the Tumour.

Myelo Cystic Tumour of the Humerus.

Fig 2.

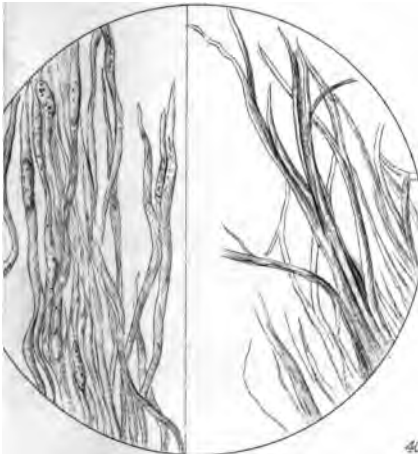


1. Fibrous tissue from the solid matter of the Tumour
400 diam.

2 Arrangement of the capillaries in the solid matter
of the Tumour

Myelo Cystic Tumour of the Femur

Fig 3. Fig 4.



3&4. Nucleated fibres and fibrous tissue
(4) from the fibro cystic portion of the Tumour.

Fig 5.



400 diam.

5 Myeloid cells and nuclear spindle shaped fibres
from the buff coloured portion of the Tumour.



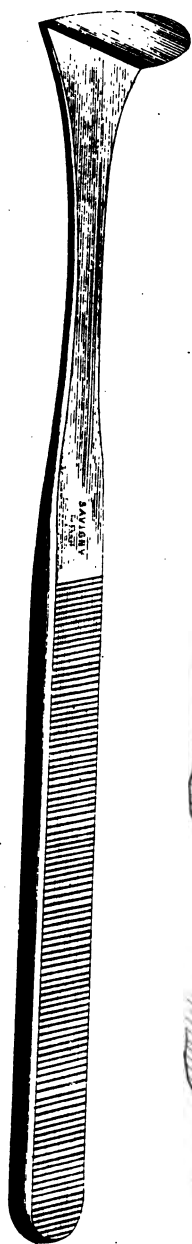


Fig 5.

CE Ford lith

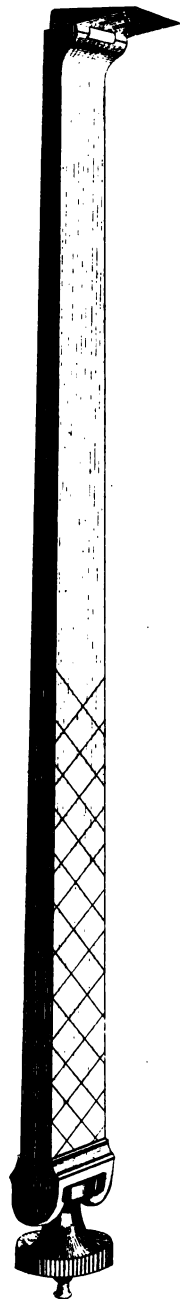
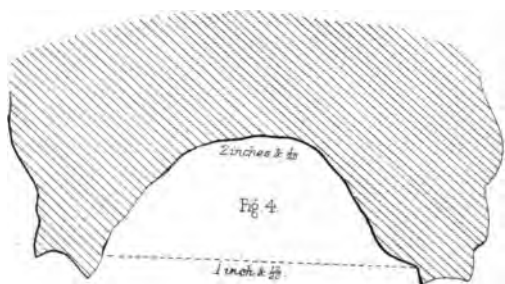
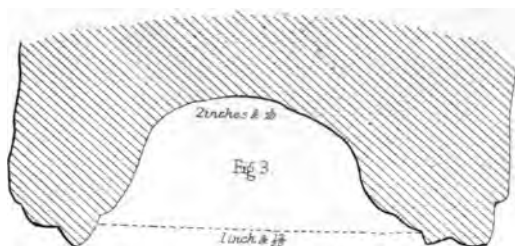
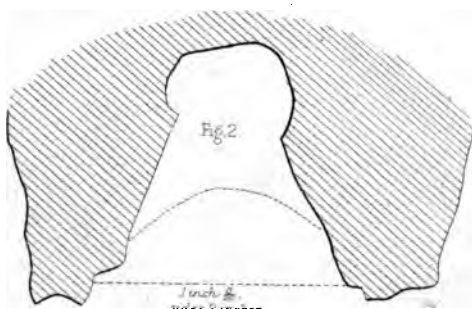


Fig 6

H West. int

H Wilson del

Phthisical persons.



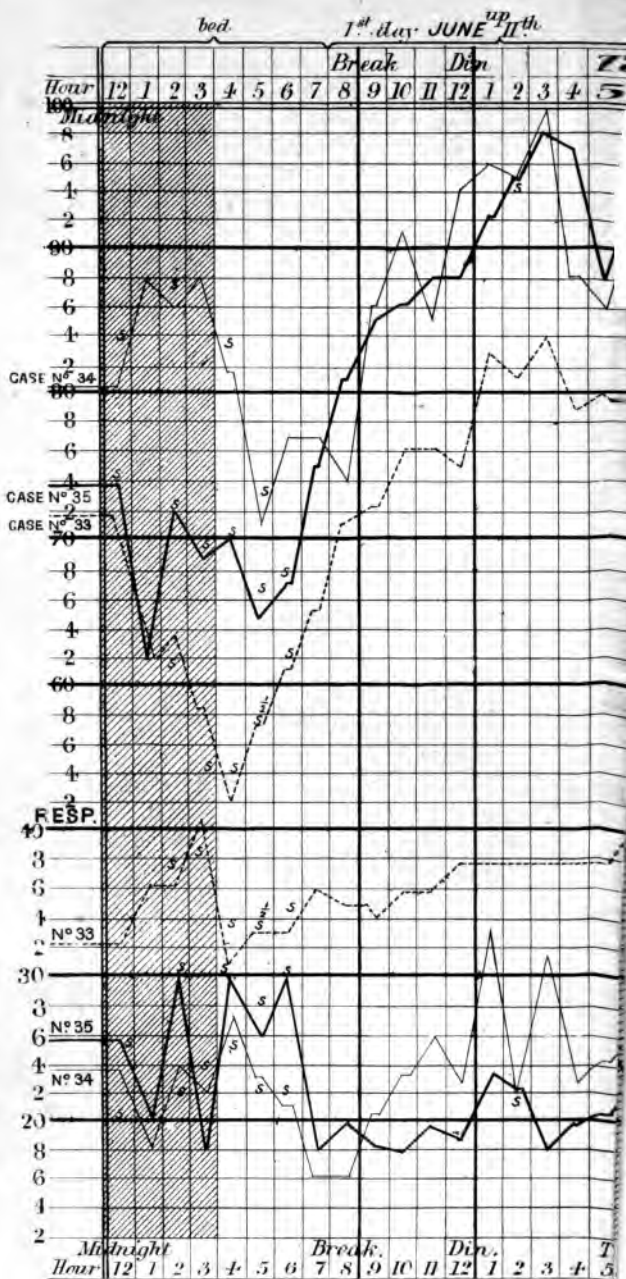




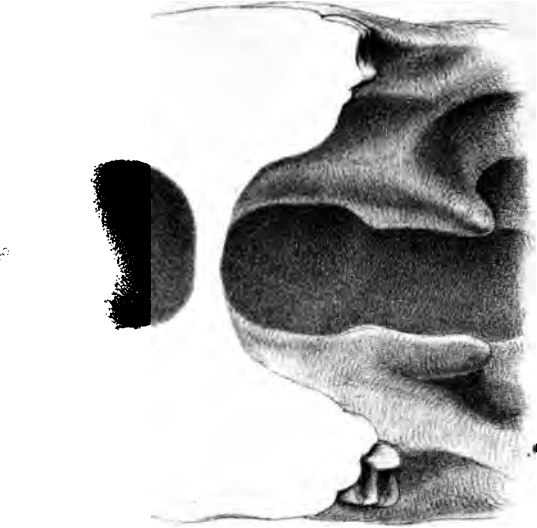
Fig. 1



W. H. Gifford, M.D.

Section to show the arch in a perfectly developed mouth.

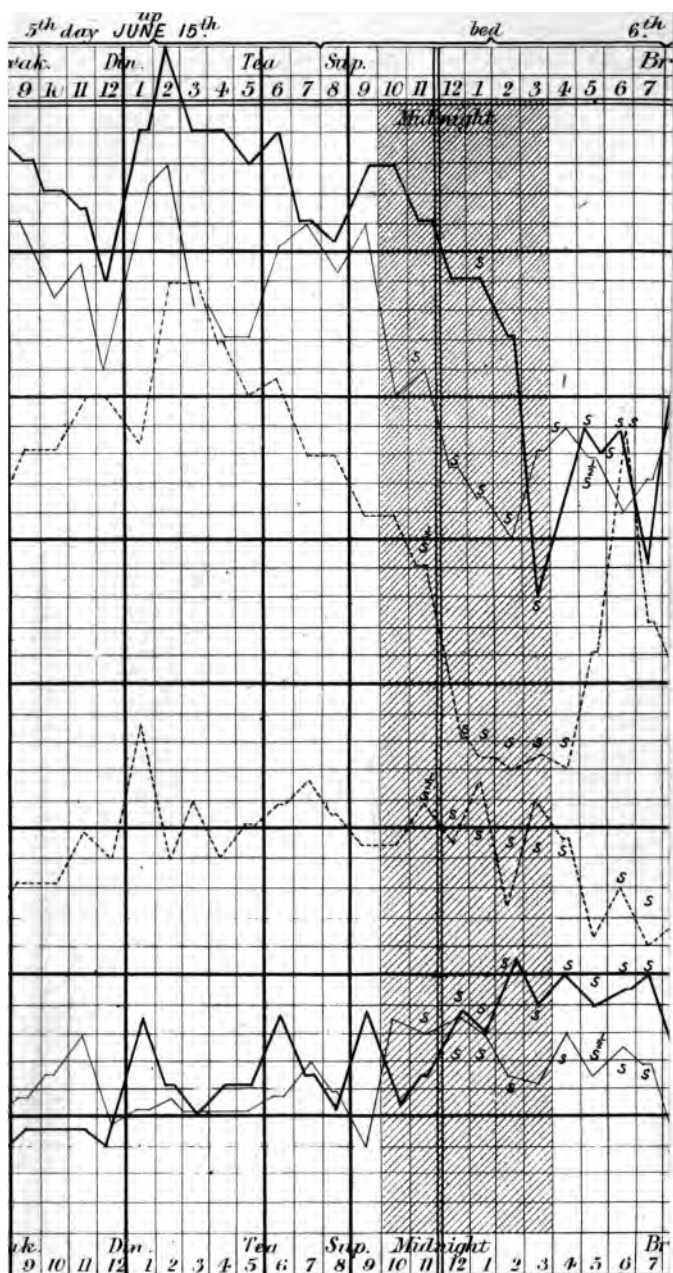
Fig. 2

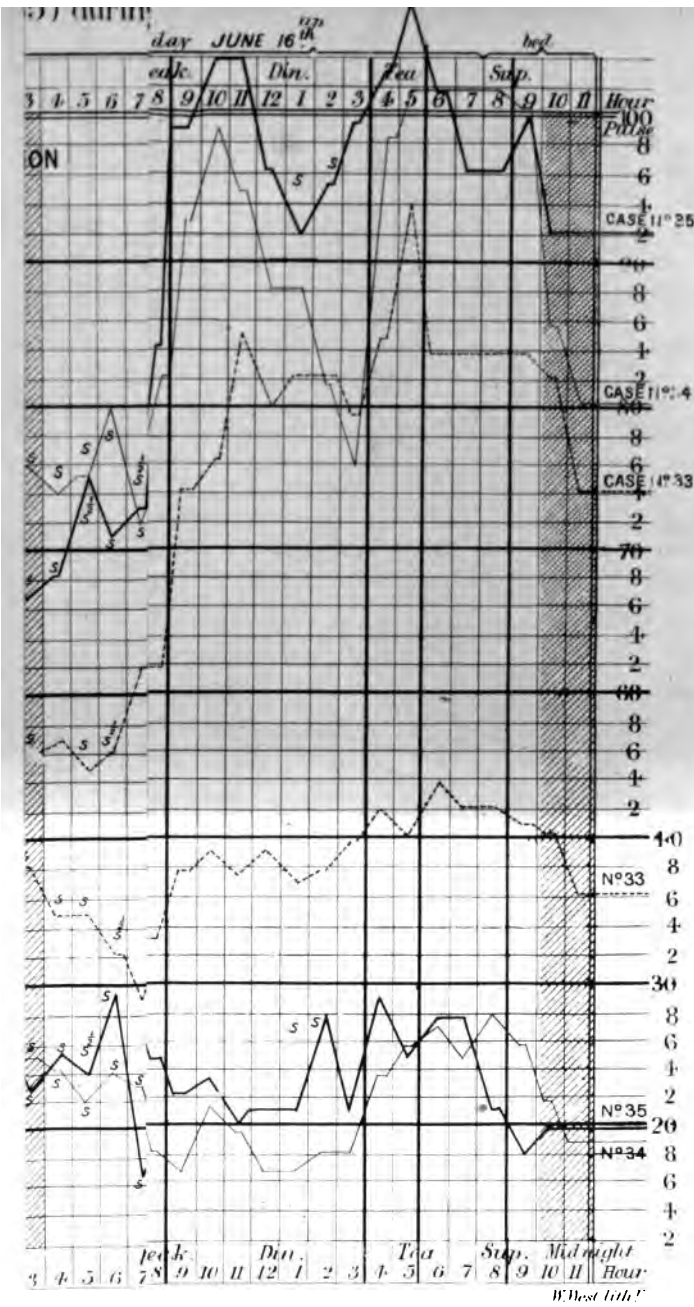


W. H. Gifford, M.D.

Section to show that the arch of the palate is almost natural when fissure is confined to the palate bones & soft palate.





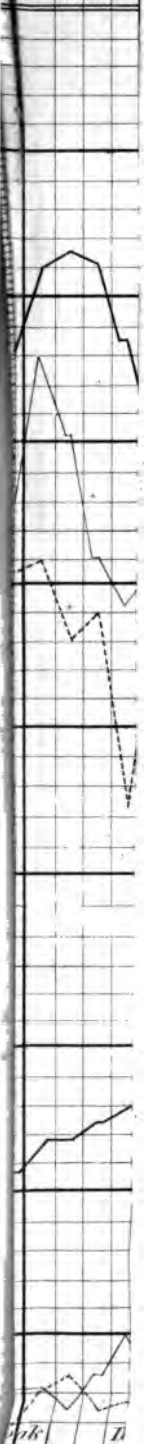


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Smith, 188

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Peak	D
9	10
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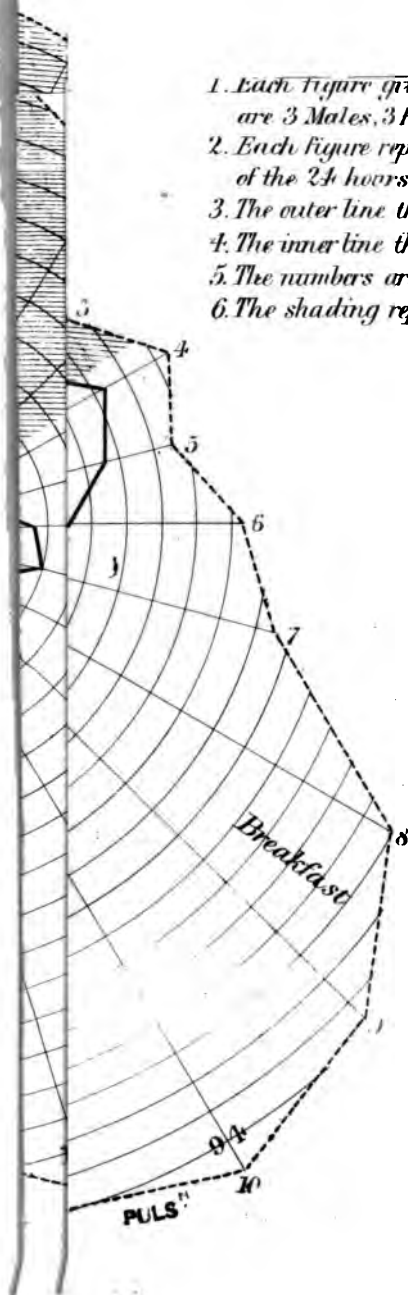




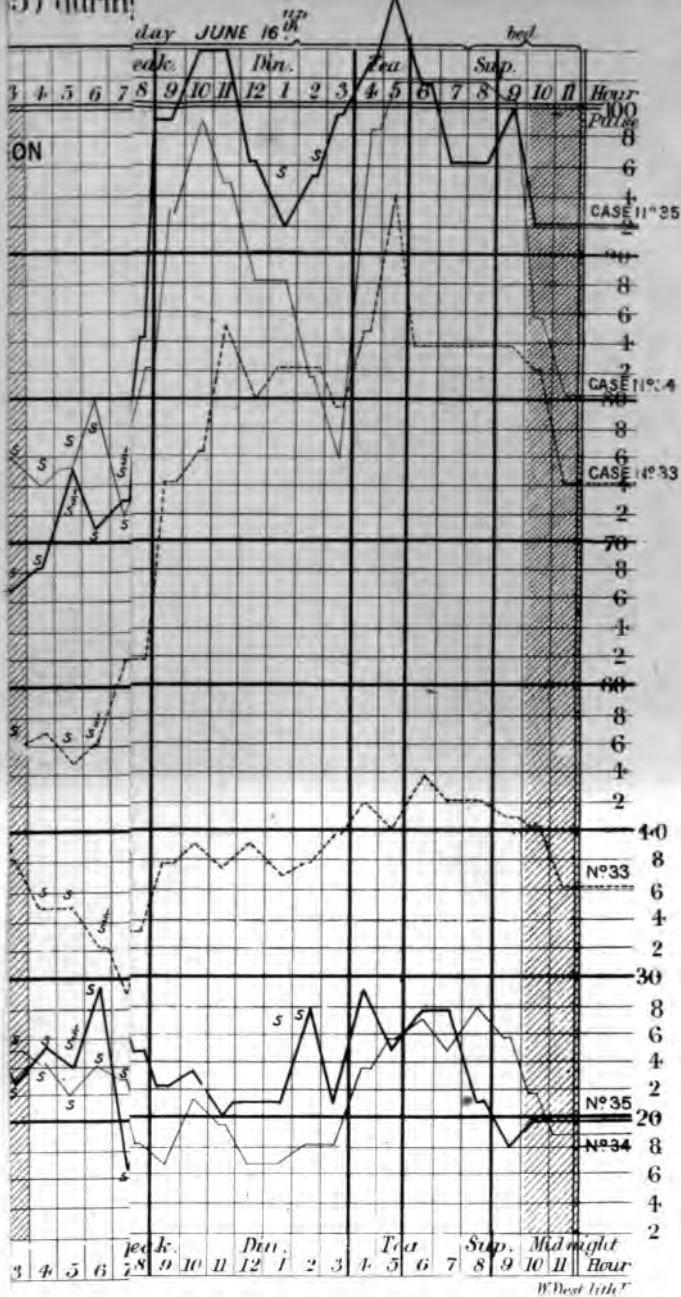




1. Each figure gives the average of a case & there are 3 Males, 3 Females & 1 Total Average.
2. Each figure represents a clock dial with a radius of the 24 hours.
3. The outer line thus represents Pulsation.
4. The inner line thus _____ " Respiration.
5. The numbers are shown on the rays & circles.
6. The shading represents darkness.

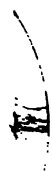


5) during



ness.

12



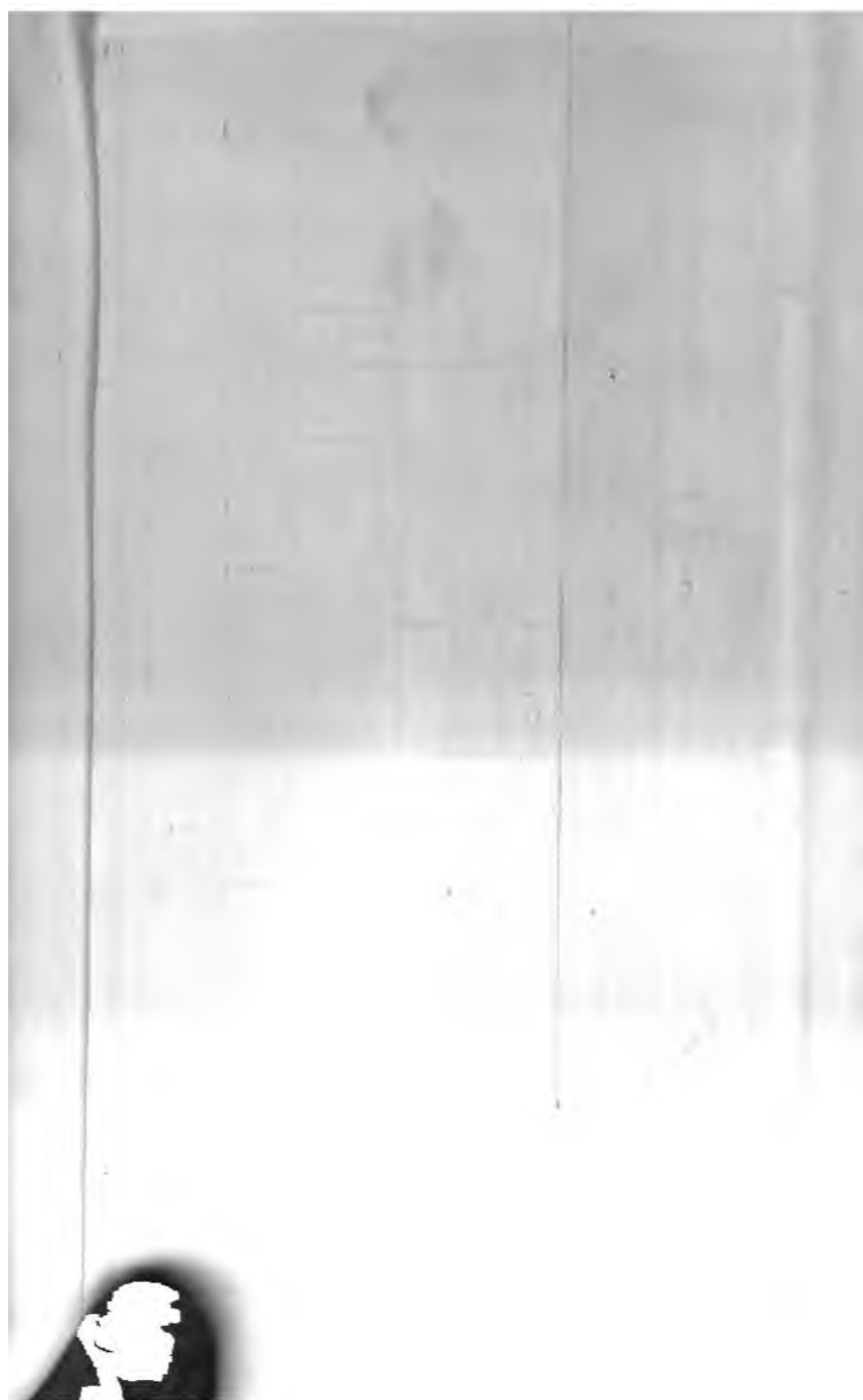
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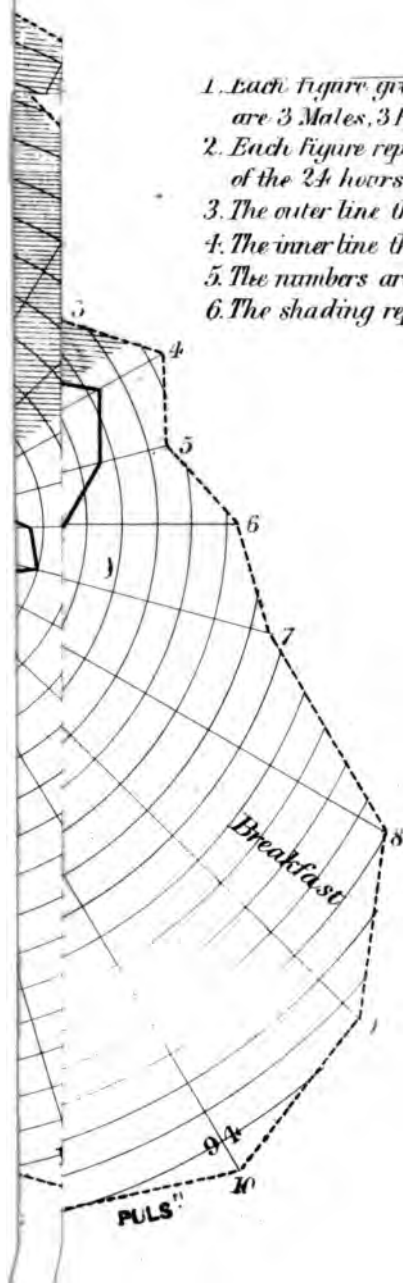
14



15



1. Each figure gives the average of a case & there are 3 Males, 3 Females & 1 Total Average.
2. Each figure represents a clock dial with a radius of the 24 hours.
3. The outer line thus represents Pulsation.
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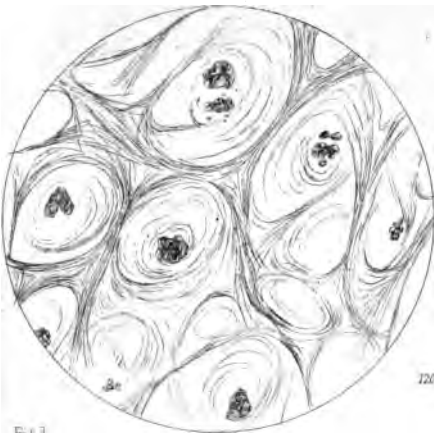


Fig 1

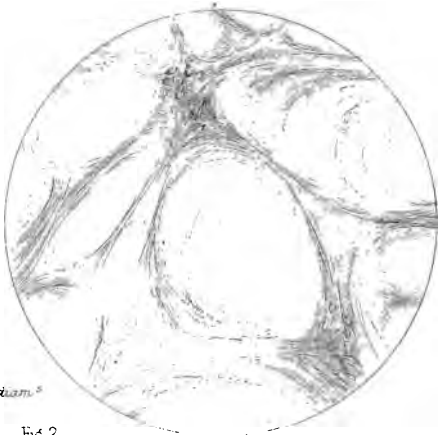


Fig 2

120 diam.^s

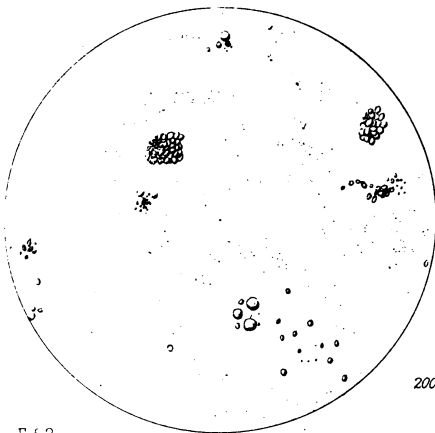


Fig 3

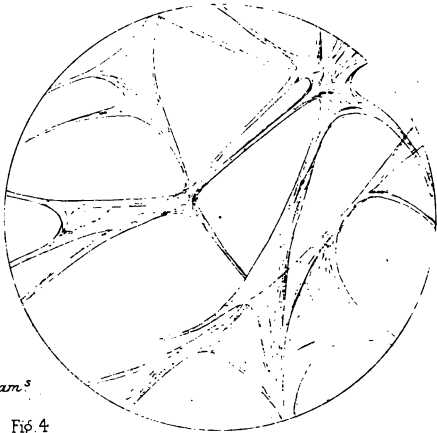


Fig 4

200 diam.^s



Fig 5

120 diam.^s



Fig 6

1. The first part of the document is a list of names and addresses of the members of the committee who have been appointed to investigate the matter.

Fig 1.

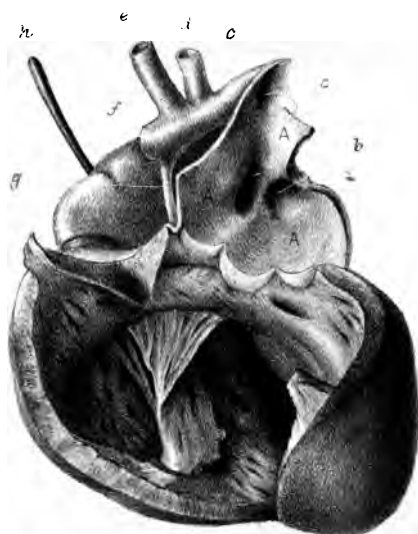
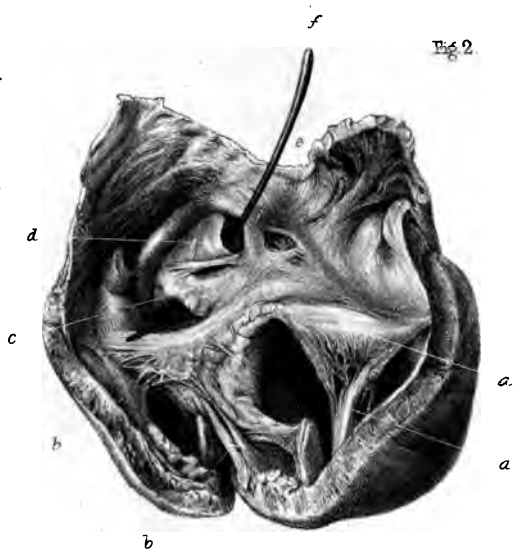


Fig 2.



2

Fig. 1.

Vol. XX.





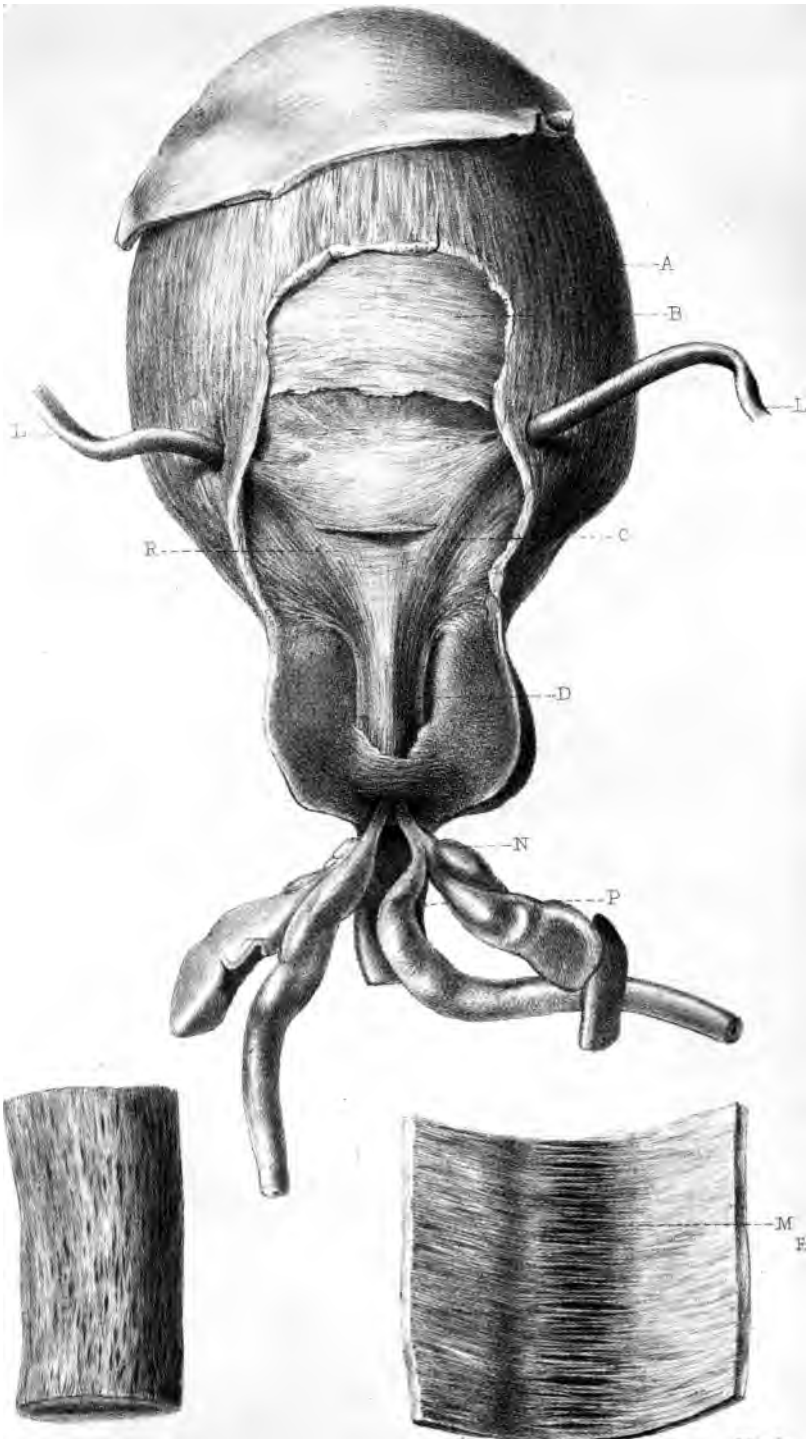


Fig. 3.



